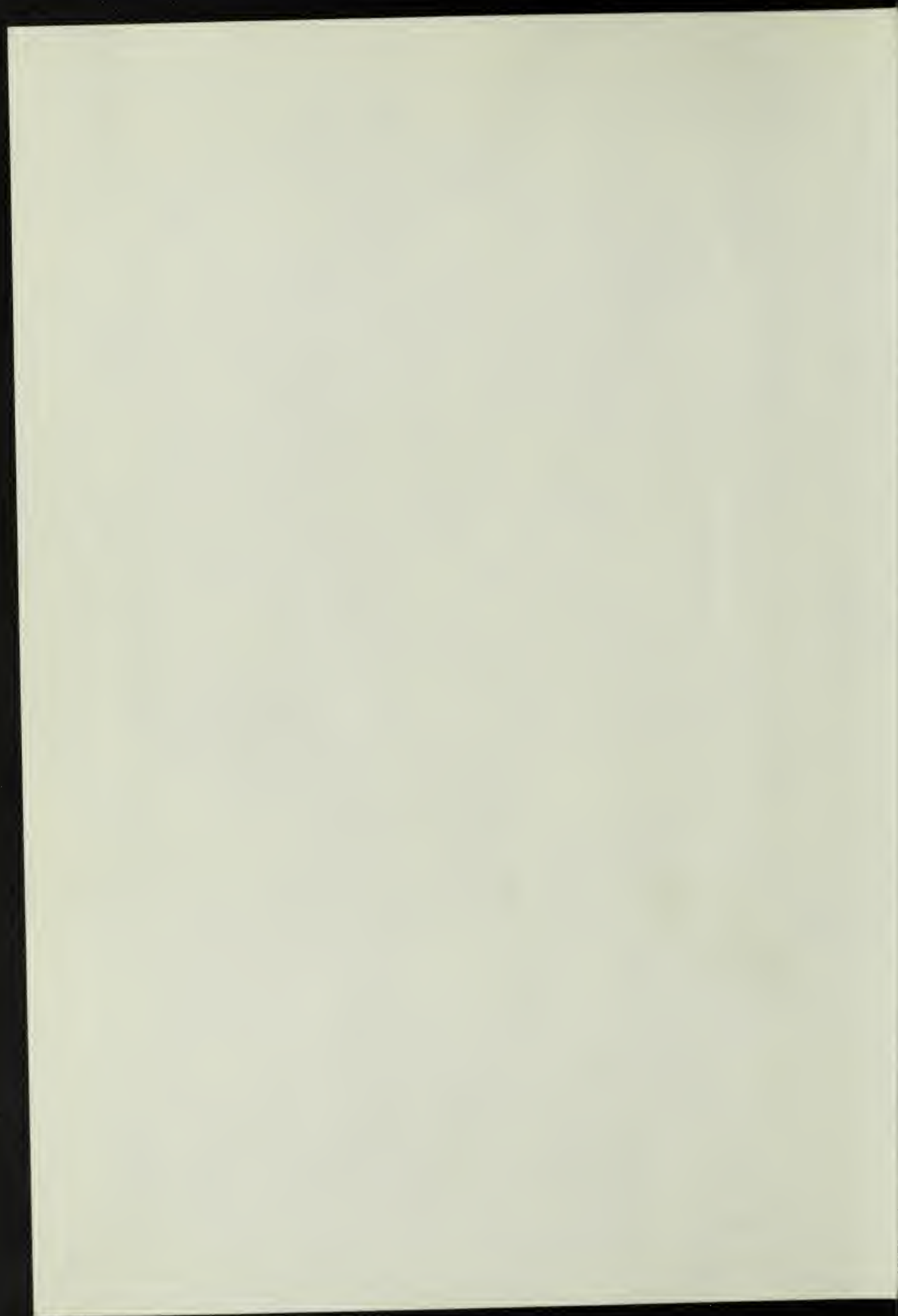




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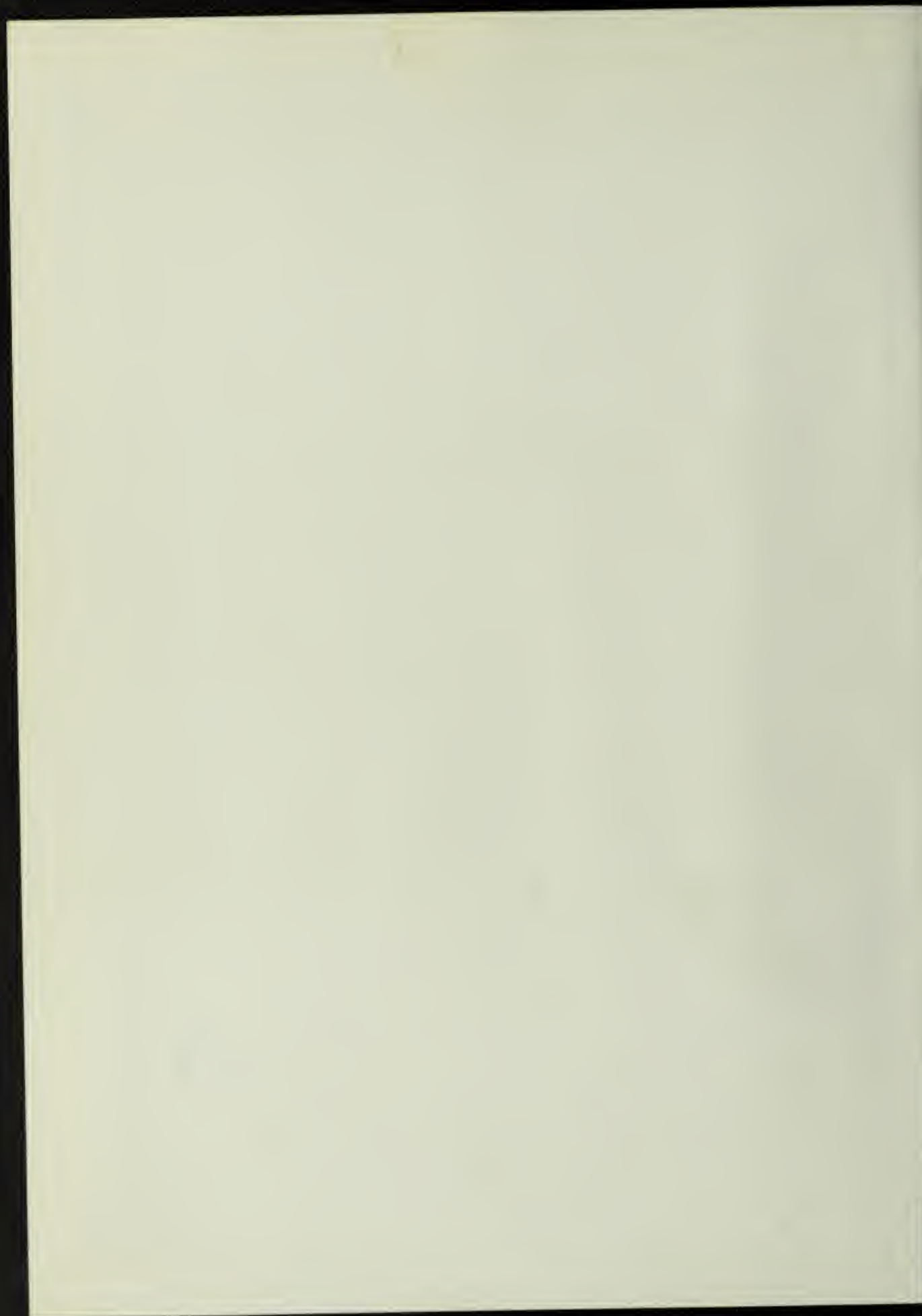












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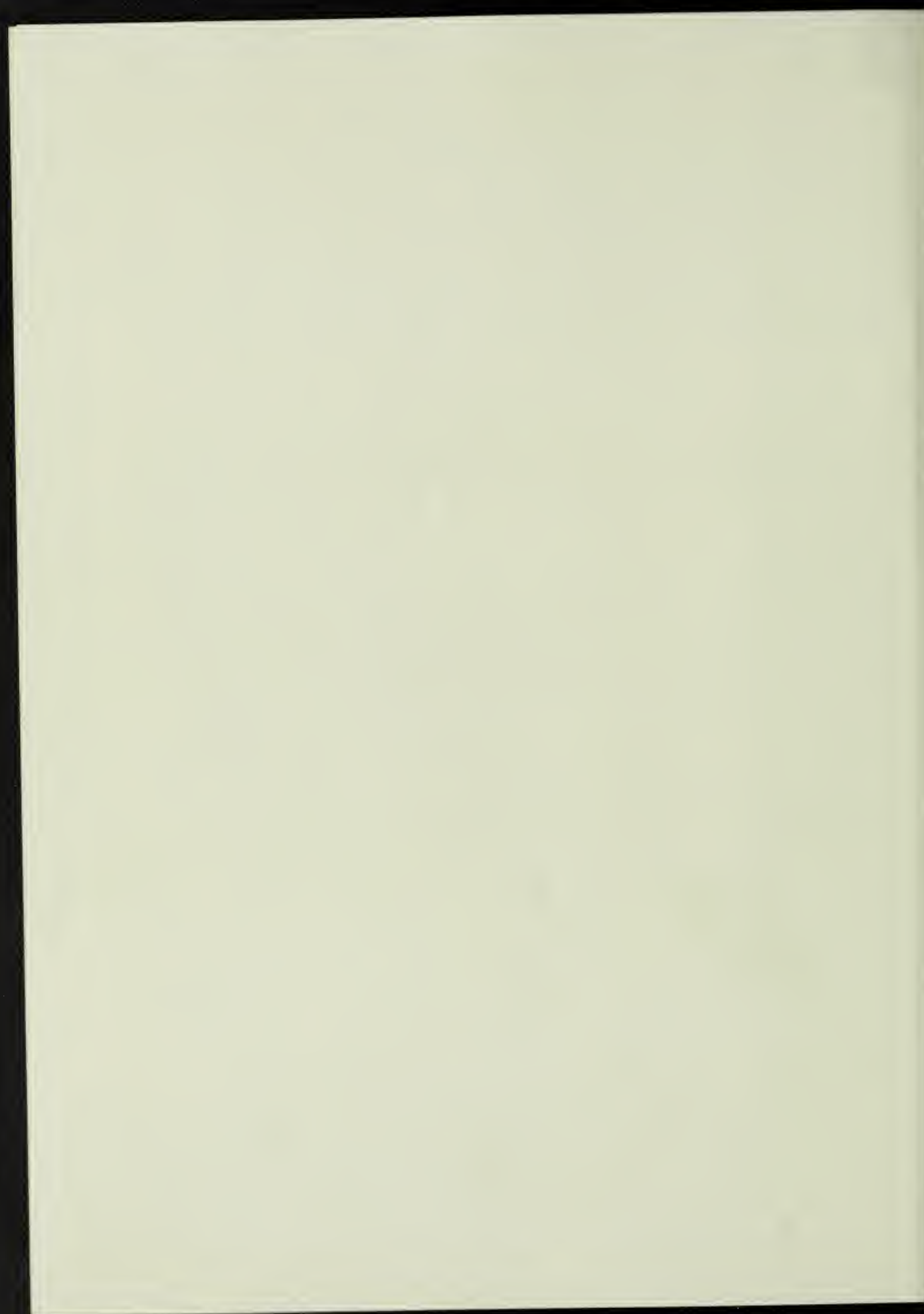
# CARCINOGENESIS ABSTRACTS

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# CARCINOGENESIS ABSTRACTS

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### GENERAL INFORMATION

CARCINOGENESIS ABSTRACTS makes available abstracts, annotations or citations of significant carcinogenesis articles collected from the current major biomedical sources of world literature. This service is provided by the National Cancer Institute through a contract with The Franklin Institute Research Laboratories for preparation of the publication, under Contract No. NOI-CP-75885 with the National Cancer Institute, U.S. Department of Health, Education and Welfare. Published and distributed by The Franklin Institute Press<sup>SM</sup>.

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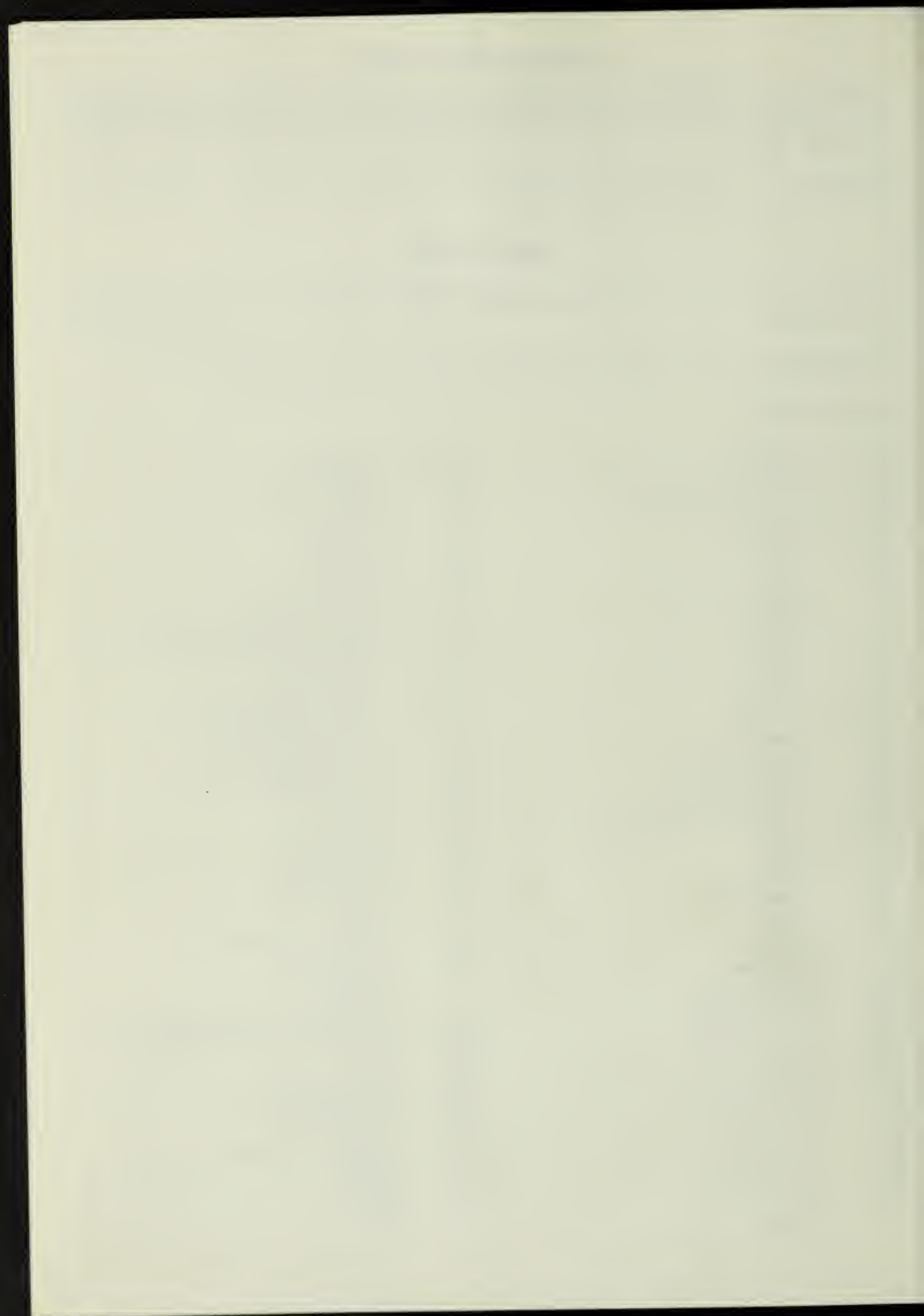
## ABBREVIATIONS

**JOURNAL** names are abbreviated according to the *List of Journals Indexed in Index Medicus, Abbreviation Listing*. If the journal is not listed in this, abbreviations are derived from the *International List of Periodical Title Word Abbreviations*.

**LANGUAGE** of the article is indicated in parentheses after the title and is represented by a three-letter code. The source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

**ABBREVIATIONS** used in abstracts:

A	angstrom(s)	mOsm	milliosmolar
ACTH	adrenocorticotrophic hormone	max	maximum
ADP	adenosine diphosphate	mEq	milliequivalent(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
approx	approximately	$\mu$ l	microliter(s)
av	average	mm	millimeter(s)
BCG	bacillus Calmette-Guerin	mo	month(s)
bid	twice daily	mol wt	molecular weight
C	degree(s) centigrade	N	normal concentration
cal	calorie(s)	NAD	nicotinamide adenine dinucleotide
kcal	kilocalorie(s)	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
Ci	curie(s)	NADPH	reduced nicotinamide adenine dinucleotidephosphate
mCi	millicurie(s)	NCI	National Cancer Institute
$\mu$ Ci	microcurie(s)	NIH	National Institutes of Health
cm	centimeter(s)	PAS	periodic acid-Schiff
CNS	central nervous system	po	orally
cpm	counts per minute	ppb	parts per billion
DNA	deoxyribonucleic acid	ppm	parts per million
ED <sub>50</sub>	median effective dose	qid	four times daily
EDTA	ethylenediamine tetraacetic acid	qod	every other day
g	gram(s)	QO <sub>2</sub>	oxygen quotient
kg	kilogram(s)	R	roentgen
mg	milligram(s)	RBC	red blood cells (erythrocytes)
$\mu$ g	microgram(s)	RNA	ribonucleic acid
Hb	hemoglobin	rpm	revolutions per minute
hr	hour(s)	sc	subcutaneous
ia	intra-arterial	sec	second(s)
id	intra-dermal	SGOT	serum glutamic-oxaloacetic transaminase
IgA	Immunoglobulin A	SGPT	serum glutamic-pyruvic transaminase
IgB	Immunoglobulin B	soln	solution
IgG	Immunoglobulin G	TCD	tissue culture dose
IgM	Immunoglobulin M	TCD <sub>50</sub>	median tissue culture dose
ILS	increased life span	tid	three times daily
im	intramuscular	UV	ultraviolet
ip	intraperitoneal	WBC	white blood cells (leukocytes)
IU	International Unit(s)	wk	week(s)
iv	intravenous	wt	weight
K <sub>m</sub>	Michaelis constant	X	times
LD	lethal dose	yr	year(s)
LD <sub>50</sub>	median lethal dose		
M	molar		
$\mu$ M	micromolar		





## REVIEW

- 77-1801 **A Unifying Biochemical Theory of Cancer, Senescence and Maximal Life Span.** (Eng.) Duchesne, J. (Dept. Atomic and Molecular Physics, Inst. Physics, Liege Univ., B-4000 Sart-Tilman, Liege, Belgium) *J Theor Biol* 66(1): 137-145; 1977.

The theory that cancer is caused by antioxidants has been extended and related to many well-defined facts, among which the most important is the discovery that malignant tumors are characterized by an abrupt drop in the level of organic free radicals. On this fundamental observation, as well as on the similar behavior of embryonic tissues, it is hypothesized that the accumulation of antioxidants is a determinant to cancer. This might be related to an unbalanced chromosome set that would then be the ultimate cause of malignancy. All this is then integrated with senescence and maximal life span in a single theory characterized by a link between oxidation and reduction, bringing together organic free radicals and antioxidants. (76 refs.)

- 77-1802 **Chemical Carcinogens.** (Eng.) Rademacher, P. (Universitat Munster, Munster, Westphalia, W. Germany) *J Chem Educ* 53(12): 757-761; 1977.

A brief review is presented of current concepts of chemical carcinogenesis. Discussion is made of the initiation and promotion of tumors; the history of the association of cancer with chemical carcinogens; the mode of action of carcinogens, including metabolism to active species; the organ specificity of N-nitroso compounds; and rules for working with carcinogens. The use of chemical carcinogens in organic syntheses can sometimes be obviated by the use of alternative reagents and synthetic routes; ie, diazomethane can be synthesized from the noncarcinogen nitrosomethyltosylamide rather than from the carcinogen nitrosomethylurea. (13 refs.)

- 77-1803 **Diazepam and Oxazepam.** (Eng.) IARC Working Group *In: IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Some Miscellaneous Pharmaceutical Substances.* International Agency for Research on Cancer (Lyon, France): Vol. 13, pp. 57-73; 1977.

Chemical and physical data; production, use, occurrence, and analysis; and available metabolic, mutagenic, toxicological, and carcinogenic data are given for diazepam (DP: 7-chloro-1,3-dihydro-1-methyl-5-phenyl-2H-1,4-benzodiazepin-2-one) and oxazepam (OP: 7-chloro-1,3-dihydro-3-hydroxy-5-phenyl-2H-1,4-benzodiazepin-2-one), a major metabolite of DP. DP is used to treat anxiety, various spastic disorders, and epilepsy and as a preanesthetic in childbirth. OP is used to treat anxiety and epilepsy. OP is carcinogenic in mice: liver-

cell adenomas were found in 3/12 male Swiss-Webster mice fed a diet containing 0.05% OP and in 8/13 male and 5/8 female mice fed 0.15% OP (the mice were fed the diet from 3- to 12 mo of age and killed at 14 mo of age). The oral LD50 of DP in mice is 278 mg/kg and that of OP is 1,540 mg/kg. DP crosses the human placenta: a significant association between maternal intake of DP during the first trimester of pregnancy and the frequency of oral clefts in the children has been reported. No data on the carcinogenicity of DP or OP in humans are available. (74 refs.)

- 77-1804 **Hycanthone and Hycanthone Mesylate.** (Eng.) IARC Working Group *In: IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Some Miscellaneous Pharmaceutical Substances.* International Agency for Research on Cancer (Lyon, France): Vol. 13, pp. 91-100; 1977.

Chemical and physical data; production, use, occurrence, and analysis; and available metabolic, mutagenic, toxicological, and carcinogenic data are reported for hycanthone (HC: 1-[2-(diethylamino)ethyl] amino-4-(hydroxymethyl)-9H-thioxanthen-9-one) and for hycanthone mesylate (HCM: the monomethane sulfonate salt of HC), which have been used as schistosomicides. HCM is carcinogenic in mice previously infected with *Schistosoma mansoni*. Of 47 *S. mansoni*-infected female CFW mice treated with a single im injection of 3 or 60 mg/kg HCM, 70% developed diffuse hepatic hyperplasia, 26% nodular benign hyperplasia, and 8.5% hepatocellular carcinomas. Of 66 similarly infected untreated mice, 29% developed diffuse hepatic hyperplasia, 15% nodular benign hyperplasia, and 1.5% hepatocellular carcinomas. The limited data were not sufficient to evaluate whether HCM is carcinogenic in non-infected animals. No data as to the carcinogenicity in man are available. Of eight patients treated with a single im injection of 3 mg/kg HC for *S. haematobium* infection, two developed severe hepatocellular injury, with the histological pattern of acute toxic hepatitis. (34 refs.)

- 77-1805 **Oxymetholone.** (Eng.) IARC Working Group *In: IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Some Miscellaneous Pharmaceutical Substances.* International Agency for Research on Cancer (Lyon, France): Vol. 13, pp. 131-139; 1977.

Chemical and physical data; production, use, occurrence, and analysis; and available metabolic, mutagenic, toxicological, and carcinogenic data are given for oxymethalone [OM: 17-hydroxyl -2- (hydroxymethylene)-17-methyl- $\alpha$ , 17 $\beta$ -androstane-3-one], a synthetic androgenic-anabolic steroid

hormone used in clinical therapy to maintain a positive nitrogen balance and in the treatment of anemias. Ten cases of liver cell tumors have been reported in young patients treated for extended periods with OM alone or in combination with other agents for aplastic anemia (5 cases), Fanconi's anemia (3 cases), or paroxysmal nocturnal hemoglobinuria (2 cases). In five of the patients, blood cysts (peliosis hepatis), hemorrhagic lesions, or hemosiderosis were found in both neoplastic and normal tissues. No metastases were seen in any patients. In two patients, the liver lesions were reported to regress after withdrawal of OM. There is insufficient evidence to conclude definitively that OM treatment caused the observed tumors. No data from animal studies are available. (28 refs.)

- 77-1806 Carcinogenic Action of Polycyclic Hydrocarbons in Animals and in Man.** (Eng.) Shabad, L. M. In: *Air Pollution and Cancer in Man. Proceedings of the Second Hanover International Carcinogenesis Meeting held in Hanover, 22-24 October, 1975.* Mohr, U.; Schmahl, D.; Tomatis, L.; Davis, W., eds. International Agency for Research on Cancer. (Lyon, France): IARC Scientific Publications No. 16 pp. 257-269; 1977.

Basic data on the carcinogenic action of polycyclic aromatic hydrocarbons (PAH) in animals and in man are presented. Benzo(a)pyrene (BP), a powerful carcinogen that is widely spread in the human environment, can be considered an indicator of PAH. Quantitative spectral-fluorescent methods were used to study the circulation of BP in the atmosphere, hydrosphere, soil, plants, animals and humans. The accumulation and degradation of PAH and the background level of BP in the environment are described. The results were used as a basis for establishing health standards and other preventive measures against cancer. (57 refs.)

- 77-1807 Microsomal Metabolism of Chemical Carcinogens in Animals and Man.** (Eng.) Marquardt, H. In: *Air Pollution and Cancer in Man. Proceedings of the Second Hanover International Carcinogenesis Meeting held in Hanover, 22-24 October, 1975.* Mohr, U.; Schmahl, D.; Tomatis, L.; Davis, W., eds. International Agency for Research on Cancer. (Lyon, France): IARC Scientific Publications No. 16 pp. 309-328; 1977.

As the first step in the interaction between chemical and cell, it has been recognized that most chemical carcinogens must be metabolically converted into reactive, electrophilic ultimate carcinogens. Ironically, the enzymes that activate carcinogens are the same microsomal, drug-metabolizing enzymes, mostly mixed-function oxidases, whose primary function is the detoxification and disposal of foreign chemicals. From the limited data available, it appears that animals and man metabolize carcinogens via very similar pathways. However, there are marked species-related and individual dif-

ferences in the basal levels of microsomal metabolism as well as in its inducibility. Therefore, existing animal data on the metabolic activation and carcinogenesis of chemicals cannot be extrapolated to man. It might be possible to detect high-risk individuals, ie, those especially endangered by exposure to environmental carcinogens due to their high rate of microsomal metabolism (eg, some cigarette smokers), by determinations of microsomal metabolism in vitro, using human WBC, or in vivo, employing safe drugs that are metabolized in a similar way to carcinogens. (100 refs.)

- 77-1808 Regulation of Drug Metabolism in Man by Environmental Chemicals and Diet.** (Eng.) Conney, A. H. (Dept. Biochemistry and Drug Metabolism, Hoffmann-LaRoche Inc., Nutley, NJ 07110) Pantuck, E. J.; Hsiao, K. C.; Kuntzman, R.; Alvares, A. P.; Kappas, A. *Fed Proc* 36(5): 1647-1652; 1977.

A review is presented of the effect of polycyclic aromatic hydrocarbons and cigarette smoking on the metabolism of drugs and carcinogens in animals and man, the effect of 2,2-bis(p-chlorophenyl)-1,1,1-trichloroethane exposure on drug metabolism in man, and the effect of certain diets (such as charcoal-broiled beef) on drug metabolism in animals and man. (65 refs.)

- 77-1809 Priorities in the Investigation of Human Health Hazards in the Plastics and Synthetic Rubber Industries.** (Eng.) Selikoff, I. J. (Mount Sinai Sch. Medicine City Univ. New York, New York, NY 10029) *Environ Health Perspect* 17: 5-11; 1976.

High priority areas for investigation of industrial health hazards include where large numbers of workers are exposed, where industrial agents are widely disseminated in the community environment, where suspect agents are in use, where agents have been in use for long periods of time, and where new chemicals are scheduled for introduction. Methods of investigation, multiple factor interactions, the responsibility of industry, and further perspectives are discussed. (33 refs.)

- 77-1810 PVC: Health Implications and Production Trends.** (Eng.) Karstadt, M. (3003 Van Ness, Washington, DC 20008) *Environ Health Perspect* 17: 107-115; 1976.

The production of poly(vinyl chloride) (PVC) includes residual vinyl chloride monomer (RVCM) and certain additives, such as stabilizers, colorants, flame retardants, and plasticizers, which may pose threats to human health. The consequences of human exposure to low levels of RVCM remain to be determined. (31 refs.)



77-1811    **Asbestos Content of Dust Encountered in Brake Maintenance and Repair (Letter to Editor).** (Eng.) Newhouse, M. L. (TUC Centenary Inst. Occupational Health, London Sch. Hygiene and Tropical Medicine, London WC1E 7HT, England) *Proc R Soc Med* 70(4): 291; 1977.

High exposure to asbestos is encountered in truck brake repair, particularly when beveling new linings. The use of vacuum brushes and vacuum funnels could control exposure in this type of work. The criteria for assessing exposure levels are discussed, including the relationship between carcinogenicity and physical properties of the fibers. (5 refs.)

77-1812    **Safety of Consumer Cosmetic Talc Products (Letter to Editor).** (Eng.) Hildick-Smith, G. (Dept. Pediatrics, Coll. Medicine and Dentistry New Jersey, Rutgers Medical Sch., Piscataway, NJ) *J Toxicol Environ Health* 2(5): 1221-1222; 1977.

Animal studies and prospective and retrospective epidemiological studies of employees milling and mining cosmetic grade talc have shown no differences in the incidence of pulmonary fibrotic disease and cancer between those exposed and unexposed controls. The available data indicate that the normal usage of cosmetic grade talc is not hazardous to health. (8 refs.)

77-1813    **Odds Are Poor That Mouse Tests Are Good Predictor of Human Cancer.** (Eng.) Coulston, F. (No affiliation given) *Chem Eng News* 55(26): 44-45; 1977.

Of more than 1,600 chemicals that produce cancer in mice, only 15 are known to cause cancer in man. Human carcinogens cause cancer in many animal species. It is suggested that the intended use of a chemical be the determining factor in legislative decisions, particularly since the benefits of most food additives outweigh the possible risks. (no refs.)

77-1814    **High Doses Can Cause Cancer by Irritation, Metabolic Disruption (Letter to Editor).** (Eng.) Martin, J. G. (No affiliation given) *Chem Eng News* 55(26): 43; 1977.

It is suggested that massive test exposure of animals to potential carcinogens such as saccharin caused irritation of the tissues, leading to a lower resistance to other carcinogens and a decrease in detoxification mechanisms. This could allow the compound being tested, or any other chemical in the environment, to cause cancer. (no refs.)

77-1815    **Idea that Irritation Causes Cancer Has Long Been Disregarded (Letter to Editor).** (Eng.) Lijinsky, W. (No affiliation given) *Chem Eng News* 55(26): 45-46; 1977.

It is possible that saccharin does not cause cancer but increases the risk of tumor development. No epidemiological studies have been done on humans to determine the increased risk of bladder or other organ tumors as a result of exposure to saccharin. Saccharin is compared to the carcinogen 2-naphthylamine, which was tested at high doses in animals and later was seen to be carcinogenic at lower doses in man. (no refs.)

77-1816    **Controversy over Whether Estrogens Cause Uterine Endometrial Cancer (Meeting Abstract).** (Eng.) Anonymous. (No affiliation given.) *Chem Eng News* 55(19): 15; 1977. (no refs.)

77-1817    **Hormone Replacement Therapy and Endometrial Carcinoma (Letter to Editor).** (Eng.) Mack, T. M. (Epidemiology and Statistics Unit, Los Angeles County/Univ. Southern California Cancer Center, Los Angeles, CA 90033) Pike, M. C. *Lancet* 1(8026): 1358; 1977.

It is emphasized that hormone replacement therapy has been connected with endometrial cancer, and that arguments dismissing the evidence of this connection are based on erroneous interpretations and treatment of epidemiological data. (5 refs.)

77-1818    **A Role for Progesterone in Breast Cancer.** (Eng.) McGuire, W. L. (Dept. Medicine, Univ. Texas Health Science Center at San Antonio, San Antonio, TX 78284) Horwitz, K. B. *Ann NY Acad Sci* 286: 90-99; 1977.

The intracellular metabolism of progesterone in breast cancer is explored, and its metabolism by the tumor itself is implicated in the progression or regression of the disease. The importance of intracellular progesterone receptors (PgR) is discussed and their role in binding injected progesterone in the normal and malignant breast is described. It is suggested that PgR might be a marker of tumors that contain endocrine receptors (ER) and, as such, a measure of hormone action. A total of 236 human mammary tumors were assayed for PgR and ER, and 64 ER-negative tumors were found. Of the ER-negative tumors, only 8% had PgR compared to 62% of the ER-positive tumors. These percentages approximate the response rate to endocrine therapy based on ER, and the

results seem to be consistent with the hypothesis that PgR can be used as a marker for ER-positive tumors. (135 refs.)

- 77-1819 **Endonuclease II of *Escherichia coli* and Related Enzymes.** (Eng.) Kirtikar, D. M.; Kuebler, J. P.; Dipple, A.; Goldthwait, D. A. In: *Fundamentals in Cancer Prevention. Proceedings of the 6th International Symposium of The Princess Takamatsu Cancer Research Fund, Tokyo, 1975*. Magee, P. N.; Takayama, S.; Sugimura, T.; Matsushima, T., eds. (Baltimore: University Park Press): pp. 349-362; 1976.

The endonuclease II of *Escherichia coli* is described. Its approx molecular wt, as determined by gel filtration, is 33,000. When examined with methyl methanesulfonate (MMS)-treated DNA, the enzyme has no divalent metal requirement, but it is stimulated approx twofold by  $Mg^{+2}$ . It is active in the presence of chelating agents such as 8-hydroxyquinoline at  $6 \times 10^{-4}$  M. EDTA inhibits the enzyme's activity 71% at  $10^{-4}$  M and 90% at  $10^{-3}$  M. No inhibition was observed with transfer RNA. The enzyme has reactive sulfhydryl groups, as indicated by its sensitivity to p-chloromercurisulfonate. It produces one double-strand break in DNA treated with MMS for every four single-strand breaks. Endonuclease II recognizes DNA treated with 7-bromomethyl-12-methylbenz[a]anthracene (7-BMMB) by hydrolyzing phosphodiester bonds and by liberating N<sup>6</sup>-7-methyl-12-methylbenz[a]anthracene (DMBA) adenine and N<sup>2</sup>-DMBA guanine. The cocarcinogen phorbol myristate acetate inhibits both the hydrolysis of phosphodiester bonds and the release of base derivatives catalyzed by endonuclease II. With both MMS-treated and 7-BMMB-treated DNA as substrates, the phorbol ester inhibits the hydrolysis of phosphodiester bonds. The ester, however, does not inhibit apurinic acid endonuclease. Endonuclease II also recognizes damage in DNA due to  $\gamma$ -irradiation. The endonuclease II preparation that is free of peaks I and II is the fraction responsible. When DNA is irradiated under nitrogen and then preincubated at 37 C in the presence of oxygen for 4 hr, there is an increase in spontaneous single-strand breaks primarily induced by alkali, as well as an increase in enzyme-sensitive sites. Endonuclease II releases 7-methyladenine and 1-methyladenine as well as 3-methyladenine and O<sup>6</sup>-methylguanine from methylnitrosourea (MNU)-treated DNA. The 3,000-fold purified endonuclease II preparation from peak II hydrolyzes the phosphodiester bonds of MMS DNA to produce 3'-hydroxyl and 5'-phosphate residues. Endonuclease II is thus active on DNA treated with MMS, MNU, 7-BMMB, and  $\gamma$ -irradiation. (17 refs.)

- 77-1820 **Perspectives on the Biology of Oncogenic Papovaviruses.** (Eng.) Fareed, G. C. (Depts. Microbiology and Immunology, Univ. California at Los An-

geles, Los Angeles, CA) *UCLA Cancer Center Bull* 4(2): 6-7; 1977.

Simian virus 40 (SV40), polyoma virus, and the human papovaviruses can induce tumors when they are injected into newborn hamsters and other susceptible animals. These viruses are also capable of transforming cells growing in vitro, and the process is believed to be analogous to tumor induction in animals. Transformed cells do not produce infectious virus, but they do carry copies of the papovavirus DNA integrated in cellular chromosomal DNA. In transformed cells, the synthesis of viral structural proteins coded for late in the viral genetic material is blocked, and only early genetic information is expressed. Physiological and genetic analyses of papovaviruses implicate expression of the virus-coded gene for the T antigen in the development of the malignant phenotype. The structure of the virus and its DNA and the multiplication of the virus are briefly discussed. (5 refs.)

- 77-1821 **Summation: Molecular Mechanisms of Gene Regulation--Session 1.** (Eng.) Levine, A. J. (Moffett Labs., Princeton Univ., Princeton, NJ 08540) *Cancer Res* 36(11): 4295-4296; 1976.

This symposium on the molecular mechanisms of gene regulation catalogs a large number of alterations in cellular gene expression in tumorigenic cells. Viral gene products act upon some central cellular mechanism to redirect the expression of large numbers of cellular gene products and phenotypes. Perhaps the best-understood system is that of simian virus 40 (SV40). A single viral gene product (A gene) appears to be required for the establishment and possibly the maintenance of the transformed state. The A gene product is a 100,000 molecular wt protein that has been identified as the viral tumor or T-antigen present in infected, transformed, and tumor cells. This product is involved in the initiation of viral and cellular DNA synthesis, the transcriptional regulation of viral genes, and the expression of cellular enzyme activities. SV40 DNA replication is initiated at a unique origin site in the genome (0.67 unit on the SV40 map), and one might expect the A protein to interact with this site. Furthermore, the 5' ends (where transcription may begin) of the polysomal messenger RNA for the early (A gene) and late (coat protein) genes each overlap the origin site of the SV40 chromosome. The SV40 A protein, residing at a single site on the virus chromosome, has the opportunity to initiate viral DNA replication, modulate the synthesis of early transcripts, and initiate (either directly or indirectly as a consequence of DNA replication) the late viral transcripts of the parental viral DNA. The A gene product may be a nuclear acidic protein or a nonhistone nuclear protein. It may be that tumor viruses have developed regulatory mechanisms for their own replication that are so closely related to the host cell that these viruses have the ability, in the end, to control the infected and the transformed cell. (1 ref.)



- 77-1822 **The Role of Committed and Uncommitted Hematopoietic Stem Cells as Targets for Rauscher and Friend Leukemia Virus.** (Eng.) Okunewick, J. P. (Viral Oncogenesis Section, Cancer Res. Unit, Clinical Radiation Therapy Res. Center, Div. Radiation Oncology, Allegheny General Hosp., 320 E. North Ave., Pittsburgh, PA 15212) *Biomedicine* 26: 152-157; 1977.

The study of Friend and Rauscher murine leukemia viruses has produced a variety of evidence regarding the nature of the target cell(s). These viruses produce leukemias with a strong erythroid component. However, they are also pancytotoxic in their action, with demonstrable effects on differentiating myeloid and thromboid cells, immunoresponsive cells, and peripheral lymphoid cells as well. In addition, a variety of factors can influence disease expression, including mouse strain, the hematopoietic cell line being observed, and the tissue microenvironment in which leukemogenesis is taking place, as well as the viral substrain itself. The data available indicate that the target cells are found among the most primitive of the hematopoietic progenitor cells of both the marrow and the spleen. However, analysis of these data suggests that the virus target is not exclusively limited to a single type of hematopoietic precursor cell. Rather, there is a closely related family of targets consisting of the uncommitted pluripotent stem cell and the committed progenitor stem cells of the erythroid, myeloid, thromboid, and immune cell lines. The evidence for each of these types of hematopoietic cells is reviewed. (54 refs.)

- 77-1823 **Intracellular and Systemic Regulation of Biologically Distinguishable Endogenous Type C RNA Viruses of Mouse Cells.** (Eng.) Aaronson, S. A. (Lab. RNA Tumor Viruses, Viral Carcinogenesis Branch, NCI, Bethesda, MD 20014) Stephenson, J. R. *Contemp Top Immunobiol* 6: 107-126; 1977.

The biological properties of endogenous mouse C-type viruses and factors that affect their expression are reviewed. There are at least three biologically distinguishable endogenous mouse C-type viruses that can be discriminated on the basis of standard host range, serological tests, and radioimmunoassays. Each endogenous mouse C-type virus examined so far can be placed into one of these three virus classes. Studies with halogenated pyrimidines and inhibitors of protein synthesis (both highly efficient inducers of endogenous mouse C-type viruses) provide strong evidence for the differential regulation of the three classes. The sera of many but not all mouse strains possess high-titer neutralizing activity specifically directed against xenotropic endogenous virus. This inhibitor has been identified as a protein, and it provides an example of a systemic mechanism for the inhibition of endogenous virus expression. The results of other studies suggest that the differentiated state of the cell may also play an important role in endogenous virus regulation. Since endogenous C-type viruses have existed in many species over a long period of evolution, some selective advantage for their pres-

ence is expected. It is speculated that they may be involved in immune surveillance mechanisms, in processes such as cellular differentiation, and in information transfer. (64 refs.)

- 77-1824 **Mammalian C-Type Oncornaviruses: Relationships Between Viral Structural and Cell-Surface Antigens and Their Possible Significance in Immunological Defense Mechanisms.** (Eng.) Schafer, W. (Max-Planck-Institut für Virusforschung, Tübingen, W. Germany) Bolognesi, D. P. *Contemp Top Immunobiol* 6: 127-167; 1977.

Several viral components and substructures have been identified, but most reactivities of special interest with regard to viral biological functions are associated with glycoprotein gp71 and, possibly, the other viral envelope constituents gp45 and p15. The p12 polypeptide is probably situated at or near the viral surface, but not on the virus exterior. The p31 and p10 polypeptides are the major constituents of the virus core, p10 being the most basic protein of the virus. The viral envelope antigens share many properties in common with gp71, such as type- and group-specific reactivities and localization on the viral and cellular membranes. At least two of the structural components on the virion surface elicit autogenous immunity in mice. Injection of mice with purified gp71 antigen results in both humoral and cellular immune responses. STU mice have been vaccinated successfully with purified Friend leukemia virus (FLV) gp71 against both FLV- and Rauscher leukemia virus (RLV)-induced leukemia. STU mice infected with FLV or RLV have also been treated successfully with anti-gp71 serum raised in goats or rabbits as a preventive measure against leukemia. (71 refs.)

- 77-1825 **Autoimmunity, Oncornaviruses, and Lymphomagenesis.** (Eng.) Hirsch, M. S. (Infectious Disease Unit, Massachusetts General Hosp., Boston, MA 02114) Proffitt, M. R.; Black, P. H. *Contemp Top Immunobiol* 6: 209-227; 1977.

Studies on the immunological activation of endogenous C-type oncornaviruses following skin-graft-rejection reactions and graft-versus-host reactions (GVHR) and the immunopathogenesis of virus-induced murine lymphoma are reviewed. Placement of histoincompatible grafts in murine skin-transplant recipients results in a vigorous immune response in regional nodes and spleens. Certain lymphocyte populations undergo blastogenesis, during which C-type viruses become activated. In the presence of immunosuppression, these viruses may replicate within other dividing target cells, and they are not eliminated by host defense mechanisms. C-type viruses are also activated in mice undergoing GVHR. Morphological changes in the spleens of animals are similar to those observed in skin-graft recipients undergoing immunosuppression. Mice infected as neonates with Moloney murine leukemia virus (MuLV-M) develop disseminated



lymphomas of thymic origin. MuLV-M-infected thymocytes may have their normal capacity to recognize self-antigen altered, so that a normal antigen(s) is recognized as foreign and MuLV-associated antigens are viewed as self. Parallel events may occur in human disorders such as Sjogren's syndrome, systemic lupus erythematosus, and myasthenia gravis. (70 refs.)

- 77-1826 **Comparative Immunology of Carcinogenesis by DNA Viruses.** (Eng.) Tevethia, S. S. (Dept. Pathology, Tufts Univ. Sch. Medicine, Boston, MA 02111) Rapp, F. *Contemp Top Immunobiol* 6: 1-69; 1977.

Carcinogenesis by DNA viruses is reviewed with emphasis on the properties of antigens associated with cells transformed by the various DNA viruses. The oncogenic potential of DNA tumor viruses in permissive and nonpermissive hosts and the effect of the immune response on tumor viruses was analyzed for papovaviruses (simian virus 40, polyoma virus, human papovaviruses, and papilloma viruses); adenoviruses; and herpesviruses (herpes simplex virus types 1 and 2, cytomegalovirus, Epstein-Barr virus, primate herpesviruses, Marek's disease virus of chickens, and herpesviruses of rabbit and guinea pigs). Those aspects of in vitro transformation by DNA viruses that deal with the malignant potential of transformed cells and their antigenicity are also examined. The properties of intracellular and cell-surface DNA virus-induced antigens are compared. Intracellular antigens induce a humoral immune response only and do not play any role in tumor rejection. Cell-surface antigens mediate the development of a cellular immune response in the host that leads to the rejection of developing tumors. (379 refs.)

- 77-1827 **Immunity to Leukemia, Lymphoma, and Fibrosarcoma in Cats: A Case for Immunosurveillance.** (Eng.) Essex, M. (Dept. Microbiology, Harvard Univ. Sch. Public Health, Boston, MA 02115) *Contemp Top Immunobiol* 6: 71-106; 1977.

The role of tumor immunity in the pathogenesis of spontaneous leukemia, lymphoma, and fibrosarcoma in cats is reviewed. Leukemias and lymphomas of outbred cats are caused by horizontally transmitted oncornaviruses, providing evidence that naturally occurring malignant tumors of outbred mammals may be caused by viruses. Studies with cats inoculated with feline sarcoma virus or feline leukemia virus (FeLV) demonstrated that an efficient humoral immune response to the feline oncornavirus-associated cell membrane antigen (FOCMA) is associated with protection from the development of lethal tumors. In a study of 43 contact-exposed healthy cats and 8 that subsequently developed leukemia, it was found that leukemic animals have poor antibody responses to FOCMA several months or years before any clinical signs of leukemia are apparent. This finding supports the

concept of immunosurveillance. Indirect evidence has also been found that FeLV can act as an immunosuppressive agent, which may provide an explanation for the large number of cats with nonneoplastic diseases that are also coincidentally viremic with FeLV. (193 refs.)

- 77-1828 **How Does T Antigen Transform Cells?** (Eng.) Weinberg, R. A. (Massachusetts Inst. Technology Center Cancer for Res., Cambridge, MA 02139) *Cell* 11(2): 243-246; 1976.

The transformation of cells by T antigens is reviewed. Several investigators have proposed that transformation may depend upon the initiation and stimulation of host cell DNA synthesis by T antigen. The T antigen protein may bind to important regulatory sites on the cellular chromosome that influence gene expression. In a virus-transformed cell, the nuclear localization of the bulk of the T antigen may reflect the search of the T antigen protein for nucleotide sequences similar to those it normally finds on the viral genome. Lacking large amounts of viral DNA with which to associate, the T antigen binds to many sites on the cellular DNA, on which it may have no direct effect. It is hypothesized that the direct interaction of early protein with cellular DNA may elicit transformation. (39 refs.)

- 77-1829 **Suppressor Cells.** (Eng.) Anonymous (Dept. Medicine, Sloan-Kettering Inst. for Cancer Res., New York, NY) *Clin Bull* 7(1): 29-33; 1977.

Progress made at the Sloan-Kettering Institute on the study of suppressor cell function at the basic level and in the analysis of various diseases is summarized. In vitro studies have shown that human lymphocytes, when stimulated with the lectin mitogen concanavalin A (Con A), produce cells that inhibit the responses of other lymphocytes from the same donor to stimulation with Con A, phytohemagglutinin (PHA), pokeweed mitogen (PWM), or allogeneic cells in mixed leukocyte culture (MLC). Suppressor cells also prevent PWM-stimulated B lymphocytes from synthesizing and secreting immunoglobulins. Animal studies have shown that suppressor cells are activated in normal mouse spleen or lymph node cell populations when cultured in the presence of a specific immune spleen cell population. Mice immunized with tumor cells develop splenic cells that suppress allogeneic responses in MLC. The cells lack thymus antigen and are glass-adherent. The derivation of the suppressor cells is discussed along with a system of cell-surface markers that enables different T cells to be separated and studied individually. The role and mechanism of action of suppressor cells in Bruton's agammaglobulinemia, lupus erythematosus, rheumatoid arthritis, and cancer are discussed. (no refs.)

- 77-1830 **Lectins.** (Eng.) Sharon, N. (No affiliation given) *Sci Am* 236(6): 108-119; 1977.

Lectins, proteins found primarily in plants, combine specifically with sugars on cell surfaces and hence bind cells together. Approx 50 lectins have been purified and characterized chemically. Studies with lectins have convinced many researchers that the cell membrane plays a central role in controlling growth, but it is not known how this control is effected. More research is needed before it is clear whether the cell-surface alterations detected with lectins are merely correlated with transformation or have a causal role in malignancy. Lectins are playing an increasing role in immunology, helping to define the mechanism by which an antigen, acting at the cell surface, specifically triggers lymphocytes to grow, mature, and proliferate and, in some cases, synthesize antibodies. Whatever the role of lectins in nature, they will undoubtedly continue to serve as an aid in the process of charting the complex landscape of cell surfaces, in clarifying the role of surface sugars in cellular behavior and growth, and in deepening the understanding of the biology of cells. (no refs.)

- 77-1831 **The Nature of the K Cell and the Role of Antibody-dependent Cell Mediated Cytotoxicity (ADCC) in the Rejection of Tumours.** (Eng.) Greenberg, A. H. (Pediatrics and Immunology, Manitoba Inst. Cell Biology, Winnipeg, R3EOV9, Canada) Wolosin, L. B. *Ann Immunol (Paris)* 128C(1/2): 485-491; 1977.

The role of antibody-dependent cell-mediated cytotoxicity (ADCC) in tumor rejection and the characterization of the K cell are considered. A null K cell found in cytolytic, cytostatic, and microcytotoxicity assays of antibody-dependent lymphocytotoxicity was identified as a nonphagocytic, nonadherent, intermediate-sized cell of lymphoidlike morphology with  $Mg^{2+}$ -independent  $C'$  receptors, lacking both T- and B-lymphocyte membrane antigens. Observations from cytotoxic assays indicate that cells other than null K cells can mediate antitumor activity via antibody, that immunoglobulin (IgG) is not the only active Ig class, that antibody may not be the only recognition molecule, and that lysis is not the only form of ADCC-induced target cell damage. In addition to the nature of the effector cell and soluble mediator, certain membrane characteristics of the target cell can also contribute to the outcome of an ADCC reaction. Tumor cells coated with equal amounts of allogeneic and syngeneic tumor-specific antibody were lysed far more effectively by alloantibody. K-cell-mediated cytostasis, however, was demonstrated with heterologous, allogeneic, and syngeneic antisera. The ability of the K cell to act independently of an adaptive immune response in the presence of a recognition molecule suggests a potential role for this effector cell in the surveillance mechanism for tumors. (29 refs.)

- 77-1832 **The Structure and Genetics of Mouse Immunoglobulins: An Analysis of NZB Myeloma Proteins and Sets of BALB/c Myeloma Proteins Binding Par-**

**ticular Haptens.** (Eng.) Hood, L. (Div. Biology, California Inst. Technology, Pasadena, CA 91125) Loh, E.; Hubert, J.; Barstad, P.; Eaton, B.; Early, P.; Fuhrman, J.; Johnson, N.; Kronenberg, M.; Schilling, J. *Cold Spring Harbor Symp Quant Biol* 41: 817-838; 1977.

A review is presented of contemporary theories of antibody diversity and of the possible constraints placed on these theories, particularly by sequence analysis of myeloma proteins and by serologic, nucleic acid hybridization, and ontogenetic data. The relationship of genealogic patterns for human complete-variable (V) regions to both the somatic mutation and germ-line theories is discussed. NZB and BALB/c myelomas appear to be distinct populations of proteins on the basis of heavy-chain class distribution, antigen-binding properties, and sequence analysis. Because of genetic differences outside the V-region genes, there are distinct internal antigenic environments in the two strains, which leads to the clonal expansion of different sets of lymphocytes in each. Differences in the N-terminal portions among the regions from myeloma proteins binding various haptens presage multiple differences throughout the remainder of the region. Data on the complete-variable region (VH) of the amino acid sequences of nine heavy chains from myeloma proteins binding the hapten phosphorylcholine are presented. Four fundamental mechanisms for generating antibody diversity are discussed: multiple germ-line genes, somatic mutation, combinatorial association, and multispecificity. (61 refs.)

- 77-1833 **Hormonal Epidemiology of Prostatic Cancer (Letter to Editor).** (Eng.) Ablin, R. J. (Div. Immunology, Cook County Hosp. and Hektoen Inst. for Medical Res., Chicago, IL 60612) *Med J Aust* 1(13): 462-464; 1977.

The possible effects of estrogen therapy on the cell-mediated immunologic responsiveness of prostatic cancer patients are discussed. Preliminary experiments indicate that estrogen suppresses phytohemagglutinin-induced mitogenesis in peripheral blood lymphocytes from patients with nonmalignant disease as well as from patients with prostatic cancer. Further investigations are needed to determine the effects of estrogen on hormone receptors which may be present on circulating lymphocytes or tumor cells and on the synthesis of a prostate-specific antigen. (14 refs.)

- 77-1834 **Immune Suppression as Related to Toxicology.** (Eng.) Vos, J. G. (Lab. Pathology, Natl. Inst. Public Health, Bilthoven, The Netherlands) *CRC Crit Rev Toxicol* 5(1): 67-101; 1977.

Studies of immune suppression by industrial compounds and environmental pollutants are reviewed. Function studies of cell-mediated immune reactions in experimental animals have demonstrated that 2,3,7,8-tetrachlorodibenzo-p-dioxin,



di-n-octyltindichloride, and di-n-butyltindichloride impair thymus-dependent immunity. Similarly, lead and cadmium increase susceptibility to bacterial endotoxin. Procedures for detecting immunosuppression in routine toxicity studies are presented. (164 refs.)

- 77-1835 Primary Pulmonary Responses to Toxic Agents.** (Eng.) Witschi, H. (Departement de Pharmacologie, Universite de Montreal, Montreal, Canada) Cote, M. G. *CRC Crit Rev Toxicol* 5(1): 23-66; 1977.

A review is presented of initial morphological changes and biochemical events associated with lung tissue damage and repair after exposure to chemicals either airborne or carried into the lung by blood. Special consideration is given to the effects of drugs on type II alveolar cells, the role of lipid peroxidation in acute or chronic lung damage, and the effects of toxic agents on the biosynthesis of macromolecules in the lung. Observations on increased lactate dehydrogenase activities in precancerous lung tissue are briefly noted. (423 refs.)

- 77-1836 Hematologic and Oncologic Implications of Alcoholism.** (Eng.) Green, J. B. (Scott and White Clinic, Temple, TX 76501) Trowbridge, A. A. *Postgrad Med* 61(5): 149-154; 1977.

The pathophysiology and clinical management of the more common hematologic and oncologic complications seen in alcoholism are reviewed. There is an unequivocal increase in the incidence of oropharyngeal, laryngeal, and esophageal carcinoma in alcoholics. Primary hepatocellular carcinoma is also more likely to develop in patients with alcoholic cirrhosis than in the general population. Abdominal pain, rapidly increasing abdominal girth, fever, and deteriorating liver function in a cirrhotic patient should always lead to the suspicion of a hepatoma. It is suggested that there may also be an increased incidence of prostatic and pancreatic carcinoma in alcoholics. Unfortunately, the diagnosis of malignant disease in the alcoholic is often delayed because of the inattentiveness of both patient and physician. (24 refs.)

- 77-1837 Parasites in the Etiology of Cancer--Bilharziasis and Bladder Cancer.** (Eng.) Elsebai, I. (Dept. Surgery, Cancer Inst., Cairo Univ., Cairo, Egypt) *CA* 27(2): 100-106; 1977.

Factors pertaining to the possible role of parasites in the etiology of cancer are reviewed, with particular emphasis on bilharziasis and bilharzial bladder cancer (BBC). BBC differs from non-BBC in many areas: age at diagnosis, site, tumor type, degree of lymph node involvement, and clinical manife-

stations. Treatment with radiotherapy is generally disappointing, because of the usually advanced stage at diagnosis and the presence of extensive fibrosis and resistant pyogenic infection. Endoscopy and conservative surgery have little or no place in the management of BBC, not only because of the advanced stage, large size, and multiplicity of tumors, but also because of the frequency of associated, generalized precancerous lesions involving the bladder mucosa. Segmental resection offers only limited palliation. Radical cystectomy is still the only modality that can provide long-term survival. Recurrence usually occurs locally in the pelvis, and it is frequently manifested during the first or second postoperative years. Metastases in the pelvic bones, lungs, and liver may also develop. (14 refs.)

- 77-1838 Morphology and Morphogenesis of Cancer of the Gallbladder and Extrahepatic Biliary Ducts.** (Rus.) Eliseev, V. V. (Lab. Chemical Carcinogens, N. N. Petrov Scientific Res. Inst. Oncology, USSR Ministry of Public Health, Leningrad, USSR) *Arkhh Patol* 39(1): 68-77; 1977.

Literature pertaining to the pathogenesis and morphology of cancer of the gallbladder and of the extrahepatic biliary ducts is reviewed. Cholecystitis was observed in 36.3% of the patients with gallbladder cancer and in 22.5% of the patients with biliary duct cancer. Gallstones were found in 63%-92.6% of the patients with gallbladder cancer but in only 22%-57% of those with extrahepatic biliary duct cancer. Biliary duct tumors may occur as endophytic or exophytic carcinomas, the latter being less invasive and less malignant than the former. Most extrahepatic biliary duct tumors have the histology of a scirrhous adenocarcinoma. Poorly differentiated carcinomas of the gallbladder are less common than well-differentiated carcinomas. Anaplastic tumors were observed in 10% of the patients with gallbladder cancer but in only 1.6% of the patients with extrahepatic biliary duct cancer. Inflammatory hyperplasias may facilitate the malignant transformation of the epithelium, and initially benign adenomatous polyps are felt to be precancerous formations. (89 refs.)

- 77-1839 Origin of the Embryonal Features of Neoplasms and of Tumor Progression.** (Rus.) Shapot, V. S. (Moscow, USSR) *Arkhh Patol* 39(1): 3-10; 1977.

Current views on the nature and mechanism of oncogenesis are summarized. Only proliferating cells can undergo malignant transformation. Immature embryonal cells are extremely sensitive to chemical carcinogens. When 6-day embryo tissue is transplanted into the testis of an adult animal, it can undergo spontaneous transformation into a teratocarcinoma. It was suggested that dedifferentiation of the cells in normally nonproliferating tissues (liver, lung, kidney) provides immature cells that serve as targets for oncogenic factors. Actively regenerating liver tissue of rats subjected to partial hepatecto-

my acquires the embryonal forms of some enzymes (pyruvate kinase) and loses the enzymes specific for this tissue (glucokinase). Benign tumors are considered an early stage of neoplastic development. All properties of malignant tumors should be subdivided into primary properties, those also present in the benign tumors, and secondary properties. The latter should be subdivided further into constitutive (invasiveness, destructive growth, systemic action on the organism) and facultative types. (45 refs.)

- 77-1840 **Risk Factors in Hodgkin's Disease.** (Eng.) Anonymous (No affiliation given) *Lancet* 1(8017): 888-889; 1977.

The concepts that Hodgkin's disease may be caused by some infectious agent and that this agent may be horizontally transmitted are discussed. It was suggested that the agent could be spread by close continued personal contact and that transmission could be direct or via a symptomless carrier. The direction of transmission is usually from young to young and rarely from old to young. Studies have shown that family members living in the same household had shorter intervals between diagnoses than similarly related family members living apart. These findings indicate the importance of some environmental factor. Common-source exposure was unlikely because of the wide differences between times of diagnosis. Investigation of family involvement showed that if one sibling developed the disease, the risk to the other siblings was increased at least seven times. Other factors that may increase the risk of Hodgkin's disease are tonsillectomy, social conditions, race, sex, age, and genetic factors. (32 refs.)

- 77-1841 **Current Concepts in the Epidemiology and Etiology of Primary Renal Cell Carcinoma.** (Eng.) Kantor, A. F. (Connecticut Cancer Epidemiology Unit, New Haven, CT) *J Urol* 117(4): 415-417; 1977.

Current concepts in the epidemiology and etiology of primary renal cell carcinoma are reviewed. Renal cell carcinoma almost always occurs in persons > 40 yr of age, especially those in the sixth and seventh decades of life. The median survival time for adults with renal carcinoma is 2.7 yr for those 35-54 yr old at the time of diagnosis, but only 1 yr for those > 65 yr old. The incidence of renal cell carcinoma in the US is estimated to be 5.6/100,000 among men and 4.1/100,000 among women. Statistics show that the incidence is consistently higher among urban dwellers. Radiation exposure and a host of chemical agents and hormones are suspected of inducing renal carcinoma in man, but the etiology of the disease remains relatively obscure. (38 refs.)

- 77-1842 **Epidemiological Review of Lung Cancer in Man.** (Eng.) Higginson, J.; Jensen, O. M. In: *Air Pollution and Cancer in Man. Proceedings of the Second Hanover International Carcinogenesis Meeting held in Hanover,*

22-24 October, 1975. Mohr, U.; Schmahl, D.; Tomatis, L.; Davis, W., eds. International Agency for Research on Cancer. (Lyon, France): IARC Scientific Publications No. 16 pp. 169-189; 1977.

The epidemiology of lung cancer is reviewed, and the usefulness of epidemiological studies in providing clues to its etiology is evaluated. Internationally, lung cancer accounts for up to 13% of all deaths in age groups > 45 yr. It is characterized by large international variations in incidence; in addition, urban-rural differences occur in many countries. Male:female ratios are high (4:1 to 5:1) but, after a rise during the first half of the century, they are now declining in a number of countries. Neither racial distribution nor migrant studies indicate that genetic factors play any important role in the etiology of lung cancer. The only possible exception is the high incidence of lung cancer among Chinese (primarily Cantonese) women. A genetic determination of individual susceptibility has been suggested, but racial differences remain to be demonstrated. The increase in lung cancer that began during the first decades of the 20th century in Europe and North America is consistent with the introduction of a carcinogen into the environment at the turn of the century. Both spatial and temporal trends of lung cancer support the hypothesis that cigarette smoking is the major etiological factor. A decrease in lung cancer incidences in certain age groups in several countries may reflect changing smoking habits and the diminishing tar content of modern cigarettes, indicating ways of prevention. Air pollution may play a secondary role as an etiological factor. The risk of lung cancer is greater in certain occupations, often because of an interaction between industrial air pollution and smoking. It is concluded that lung cancer is largely a problem of modern society and that the epidemiological results are consistent with an etiological role of preventable environmental factors. (50 refs.)

- 77-1843 **The Multistage Theory of Carcinogenesis (Letter to Editor).** (Eng.) Hakama, M. (Finnish Cancer Registry, Helsinki, Finland) *Int J Cancer* 19(5): 730-731; 1977.

The application of the multistage carcinogenesis theory to study the age distribution of acute leukemia, Hodgkin's disease, and lung cancer is defended. (1 ref.)

- 77-1844 **The Multistage Theory of Carcinogenesis: Comment on the Letter by Dr. Moolgavkar (Letter to Editor).** (Eng.) Peto, R. (Oxford Univ., Oxford, England) Doll, R. *Int J Cancer* 19(5): 731; 1977.

It is proposed that, in spite of the lack of strong epidemiologic evidence for the multistage theory of carcinogenesis, approximation for multistage models could be applied effectively to cellular transition rates of 0.1/cell/yr. Factors that may invalidate the use of national lung cancer incidence data as



evidence of a true age-specific mode in rates are summarized. (1 ref.)

- 77-1845 **Chemical Changes in Neoplastic Cell Membranes.** (Eng.) Warren, L.; Buck, C. A. In: *Membranes and Disease*. Bolis, L.; Hoffman, J. F.; Leaf, A., eds. (New York: Raven Press): pp. 173-181; 1976.

Changes in membrane lipids, glycolipids, proteins, and glycoproteins when cells become malignant are reviewed. At present, comparative data on the neutral and phospholipids of normal and malignant cells are not extensive. No specific tumor lipids or glycolipids have yet been found. Human and mouse leukemic cells contain significantly less unesterified cholesterol than their normal counterparts. The drop in cholesterol content and the associated increase of membrane fluidity have been considered an important part of malignant change. In some studies, the malignant cells contained more glycolipids with shorter carbohydrate chains (reduced sialic acid content) and less with longer chains, leading to the generalization that malignant cells do not complete glycolipid carbohydrate synthesis. Numerous quantitative changes have been found in the amounts of protein and glycoprotein in various membrane systems when cells become malignant, but the significance of these changes has not been determined. (68 refs.)

- 77-1846 **On the Etiology and Prevention of Cancer in the Gastrointestinal Tract.** (Eng.) Weisburger, J. H.; Fiala, E.; Narisawa, T.; Reddy, B. S. In: *Fundamentals in Cancer Prevention. Proceedings of the 6th International Symposium of The Princess Takamatsu Cancer Research Fund, Tokyo, 1975*. Princess Takamatsu Cancer Research Fund. (Tokyo): pp. 125-142; 1976.

Current views on the etiology of gastrointestinal cancers are discussed. In America and Western Europe, cancer of the esophagus is associated with smoking plus heavy drinking. In parts of Asia and Africa, this association is not found. Rats fed a diet supplemented with 1,4 dinitrosopiperazine (DNP) developed papillomas and carcinomas of the esophagus in 24/32 cases. Feeding low-vitamin A diet or diets containing phenobarbital (PB), chrysene, or  $\beta$ -naphthoflavone (NF) had little effect in the induction of this cancer. Gastric cancer occurs more frequently in populations eating diets high in carbohydrates, low in micronutrients and containing high levels of nitrite. Cancer of the colon was induced in animals by the intrarectal instillation of alkyl nitrosamides or the administration of cycasin, or 1,2-dimethylhydrazine (DMH). Rats fed diets supplemented with both DMH and PB showed an increased

multiplicity and incidence of distal colon tumors; DMH plus NF gave similar results. DMH, PB, and NF increased the incidence and multiplicity of duodenal tumors. Populations eating a high fat, meat diet have increased risk of colon cancer. (75 refs.)

- 77-1847 **Cyclic Nucleotides, Thioldisulfide Status of Proteins, and Cellular Control Processes.** (Eng.)

Rebhun, L. I. (Dept. Biology, Univ. Virginia, Charlottesville, VA) Miller, M.; Schnaitman, T. C.; Nath, J.; Mellon, M. *J Supramol Struct* 5(2): 199-219; 1977.

Literature on the effects of cyclic nucleotides on cell division, cell shape, cell adhesion, and cell motility was reviewed. These effects were found to vary depending on the cells selected and the conditions under which they were used, which suggests that the effects must be indirect and could only be superimposed on whatever fundamental mechanisms exist in cells for control of the four selected characteristics. Previous studies designed to examine the inhibitory action of methylxanthines on the development of sea urchin eggs, which were based on the idea that the methylxanthines acted through cyclic nucleotides, had to be abandoned. It has since been found that methylxanthines inhibit the activation of glutathione reductase (GR) and that glutathione-oxidizing agents act as mitotic inhibitors. Tubulin polymerizability, NAD-kinase activity, and a mitotic apparatus-associated  $\text{Ca}^{2+}$ -ATP-ase were also inhibited by oxidation of some of their sulfhydryls, but they were activated by reduction of the resulting disulfides. These results suggest that it is the enzymatic uses to which glutathione is put that are of importance as the control system in normal cells and that control under physiological conditions is through GR rather than through regulation of GR levels. (109 refs.)

- 77-1848 **Doubt Concerning the Effectiveness of the Present Strategy in Screening for Cervical Carcinoma.** (Dut.) Sturmans, F. (Hoofd Afdeling gezondheids-voorlichting en -opvoeding, Gemeentelijke Geneeskundige en Gezondheidsdienst, Rotterdam, Netherlands) Valkenburg, H. A.; Burema, L. *Ned Tijdsch Geneesk* 120(28): 1191-1197; 1976.

Studies of mortality statistics have not proved the effectiveness of screening for cancer of the uterine cervix. This lack of proof may be due to the fact that screening is on a voluntary basis and may thus reach only the low-risk categories. (15 refs.)

## CHEMICAL CARCINOGENESIS

77-1849 Aldehyde Dehydrogenase in 2-Acetamidofluorene-induced Rat Hepatomas: Ontogeny and Evidence That the New Isoenzymes Are Not Due to Normal Gene De-repression. (Eng.) Lindahl, R. (Dept. Biology, Univ. Alabama, University, AL 35486) *Biochem J* 164(1): 119-123; 1977.

Aldehyde dehydrogenase (AD) in hepatomas induced by 2-acetylaminofluorene (AAF), 4-dimethylaminoazobenzene, and ethionine in Sprague-Dawley rats exists as three major and several minor isoenzymes that are not observed in any normal adult tissues. Investigation of the properties of normal AD throughout ontogeny demonstrates that the fetal enzyme is identical to the normal adult species. At no time does normal rat liver AD, by any of the criteria used (gel electrophoresis, gel isoelectric focusing, and immunochemistry) appear identical with or even similar to the AD in AAF-induced hepatomas. It is concluded that if carcinogen-induced gene derepression is responsible for the hepatoma-specific AD phenotype, the newly derepressed genes are not those normally expressed during pre- or postnatal liver development or in some other normal adult tissue. The possibility that the new AD species arises from posttranslational modification of normal AD due to carcinogen-induced alterations in other components of the hepatic metabolic machinery cannot be excluded. (22 refs.)

77-1850 The Uptake and Secretion of 2-Acetylaminofluorene by the Rat and Dog Prostate. (Eng.) Smith, E. R. (Dept. Pharmacology, Univ. Massachusetts Medical Sch., Worcester, MA 01605) Hagopian, M.; Reister, H. C. *Toxicol Appl Pharmacol* 40(2): 185-191; 1977.

The uptake and secretion of 2-acetylaminofluorene (2-AAF) by the rat and dog prostate were studied. In unanesthetized rats examined 4-140 hr after the ip administration of 1.8 mg/kg of <sup>14</sup>C-2-AAF, radioactivity was detected in the plasma, dorsal and ventral lobes of the prostate, liver, kidney, testes, and pancreas. Plasma radioactivity was max at 4 hr after treatment. In experiments with anesthetized rats, in which prostatic radioactivity was determined 25-29 hr after the ip administration of 2 mg/kg of <sup>14</sup>C-2-AAF, radioactivity was detected in the prostatic secretion of all rats examined. Three unanesthetized dogs with surgically prepared fistulas were given ip doses of 0.16-0.25 mg/kg of <sup>14</sup>C-2-AAF. Two dogs were followed for 6 hr and the third was followed for 166 hr. In all three dogs there was a rapid elevation in plasma radioactivity, followed by a gradual decline and the appearance of radioactivity in the prostatic fluid. The prostate glands of the two dogs examined 6 hr after treatment were radioactive. The results indicate that 2-AAF and/or its metabolites enter the prostate gland and its secretions in both the rat and the dog. (11 refs.)

77-1851 Inhibitory Effects of Selenium on the Mutagenicity of 2-Acetylaminofluorene (AAF) and AAF Derivatives. (Eng.) Jacobs, M. M. (Dept. Biochemistry, Univ. Texas System Cancer Center, 6723 Bertner Ave., Houston, TX 77030) Matney, T. S.; Griffin, A. C. *Cancer Lett* 2(6): 319-322; 1977.

The potential inhibitory effects of selenium on the mutagenicity of 2-acetylaminofluorene (AAF) and two of its derivatives were investigated using the *Salmonella typhimurium* TA 1538 bacterial tester system. Graded decreases in mutagenicity with increasing Se concentrations were noted for each of the three mutagens tested. Se in concentrations of 4-40 mM reduced the mutagenicity of AAF to 80%-65% of its original value. Concentrations of 0.4-40 mM reduced the mutagenicity of N-hydroxy-2-acetylaminofluorene (N-OH-AAF) to as little as 28% of its original value, and 0.1 to 20 mM reduced the mutagenicity of N-hydroxyaminofluorene (N-OH-AF) to 61% of its original value. The molar ratios of Se to mutagen that yielded the largest decreases were approx 10:1 for Se:AAF, 10:1 for Se:N-OH-AAF, and 300:1 for Se:N-OH-AF. These data suggest that Se may be important as an inhibitor of the metabolic activation of AAF and N-OH-AAF. Se may also deter mutagenic events ascribed to the activated compound, N-OH-AF, although higher molar ratios of Se: mutagen are required. (15 refs.)

77-1852 Effect of Dietary Fats on the Metabolic Activation of Chemical Carcinogens (Meeting Abstract). (Eng.) Castro, C. E. (Texas Tech Univ. and Texas Tech Univ. Sch. Medicine, Lubbock, TX 79409) Felkner, I. C.; Yang, S. P.; Sproat, H. F. *Fed Proc* 36(3): 1117; 1977. (no refs.)

77-1853 Characterization of 9-Hydroxyacetylaminofluorene: A New Metabolite of 2-Acetylaminofluorene (2-AAF) In Rat Liver Microsomes (Meeting Abstract). (Eng.) Feller, D. R. (College Pharmacology, Ohio State Univ., Columbus, OH 43210) Son, O. S.; Miller, D. D. *Pharmacologist* 18(2): 160; 1976. (no refs.)

77-1854 Distribution of Thymine Dimers and Acetylaminofluorene in Euchromatin and Heterochromatin (Meeting Abstract). (Eng.) Tomura, T. (UCLA Center for Health Sciences, Los Angeles, CA 90024)



Senko, R. A.; Van Lancker, J. L. *Fed Proc* 36(3): 1079; 1977. (no refs.)

77-1855 **Microsomal Oxidation of Arylamides (Meeting Abstract).** (Eng.) Kaplan, E. (Univ. Minneapolis, Minneapolis, MN 55417) Gutman, H. R. *Fed Proc* 36(3): 844; 1977. (no refs.)

77-1856 **Thioacetamide-induced Hepatic Necrosis. I. Involvement of the Mixed-Function Oxidase Enzyme System.** (Eng.) Hunter, A. L. (Dept. Pharmacology, E. Carolina Univ., Greenville, NC 27834) Holscher, M. A.; Neal, R. A. *J Pharmacol Exp Ther* 200(2): 439-448; 1977.

The effects of treating Sprague-Dawley rats in vivo with thioacetamide (TA) and thioacetamide sulfine (TAS) on several hepatic mixed-function oxidase (MFO) activities and the effects of experimental manipulation of MFO activities upon liver necrosis induced by TA and TAS were investigated. The activities of hepatic aniline hydroxylase 24 hr after the ip administration of 0, 0.63, 1.25, and 2.50 millimoles (mmol)/kg of TA were 0.25, 0.18, 0.16, and 0.21 unit (U), respectively. The activities after 0.63, 1.25, and 2.50 mmol/kg of TAS were 0.13, 0.08, and 0.07 (U), respectively. Similarly, the activities of aminopyrine demethylase after the same doses of TA were 3.45, 2.92, 2.48, and 3.17 U, respectively, and after TAS they were 2.47, 1.84, and 1.61 U. Thus, TAS inhibited the MFO enzymes to a greater extent than TA. TAS also produced more severe centrilobular hepatic necrosis than equivalent doses of TA. Stimulation of MFO activity by pretreatment with phenobarbital enhanced the degree of necrosis produced by both TA and TAS. Inhibitors of MFO (pyrazol, SKF 525A, and cobaltous chloride) decreased the degree of necrosis. It is concluded that both TA and TAS are activated by MFO enzymes to potent hepatotoxic compounds. It is suggested that TA ( $\text{CH}_3\text{CSNH}_2$ ) is first metabolized to TAS ( $\text{CH}_3\text{CSO}_2\text{NH}_2$ ), which is subsequently metabolized further to thioacetamide sulfene ( $\text{CH}_3\text{CSO}_2\text{NH}_2$ ). (12 refs.)

77-1857 **Effect of Portacaval Shunt on Hepatocarcinogenesis in the Rat (Meeting Abstract).** (Eng.) Ricco, J. B. (Laboratoire de Chirurgie Experimentale, Inserm U-17, Hopital Paul Brousse, Villejuif, France) Franco, D.; Morin, J.; Bismuth, H. *Digestion* 15(3): 234; 1977. (no refs.)

77-1858 **Metabolic Activation of a Hepatic Carcinogen, Safrole, by Rat Adrenal Tissue.** (Eng.) Dumas, J. (Laboratoire de Biochimie des Interactions cellulaires,

ERA CNRS 267, Universite de Dijon, 21004 Dijon Cedex, France) Maume, B. F. *C R Soc Biol* 171(1): 108-114; 1977.

The metabolism of  $^{14}\text{C}$ -1'-labeled safrole (1), a hepatocarcinogen, was studied in vivo and in vitro in the adrenal tissue of male Sprague-Dawley rats. The metabolites identified 30-100 min after ip injection of safrole were dihydro-2', 3'-dihydroxy-2', 3'-safrole, eugenol, and allyl-4-dihydroxy-1,2-benzene. The metabolites found after incubation of safrole with adrenal tissue homogenates for 2 hr were hydroxy-1'-safrole (2), allyl-4-dihydroxy-1,2-benzene (3), hydroxy-3'-safrole (4), and epoxy-2',3'-safrole (5). The metabolites found after incubation for 6 hr were compounds 2, 3, 4, an unidentified hydroxysafrole (6), and dihydro-2',3'-dihydroxy-2,3'-safrole (7). The findings indicate the existence of an epoxide-diol pathway along with demethylase and hydroxylase activities, suggesting that the adrenal cortex may participate in the metabolic activation of safrole. (17 refs.)

77-1859 **Inhibition by 3'-Methyl-4-dimethylaminoazobenzene of In Vitro Cell-free Protein Synthesis: Possible Involvement of an Electrophilic Metabolite.** (Eng.) Labuc, G. E. (Dept. Pathology, Melbourne Univ., Parkville, Victoria, 3052, Australia) Madsen, N. P. *Biochem Pharmacol* 26(10): 929-934; 1977.

The hepatocarcinogen 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB: 0.12 mM) inhibited protein synthesis 37.4% in a cell-free system using a post-mitochondrial supernatant from rat liver in the presence of NADP+. The inhibition was decreased by treatment of the postmitochondrial supernatant with Triton X-100, increased by pretreatment of the rats with phenobarbitone, and decreased by the addition of the nucleophiles (GSH reduced glutathione) and L-cysteine. There was no correlation between the inhibition effected by a series of azo dyes (3'-Me-DAB, 2'-Me-DAB, 2-Me-DAB, and DAB) on protein synthesis and their respective carcinogenic potencies. The addition of NADP+ alone to the postmitochondrial supernatant stimulated protein synthesis by > 100%. (50 refs.)

77-1860 **Summation Effect of N-Butyl-N-(4-hydroxybutyl)nitrosamine, N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide, N-2-Fluorenylacetylamide, and 3,3'-Dichlorobenzene on Urinary Bladder Carcinogenesis in Rats.** (Eng.) Tatematsu, M. (First Dept. Pathology, Nagoya City Univ. Medical Sch., 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya 467, Japan) Miyata, Y.; Mizutani, M.; Hananouchi, M.; Hirose, M.; Ito, N. *Gann* 68(2): 193-202; 1977.

The effects of the sequential po administration of 0.01% N-butyl-N-(4-hydroxybutyl)nitrosamine (BBN), 0.15% N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT), 0.025% N-2-fluorenylacetylamide (2-FAA), and 0.3% 3,3'-

dichlorobenzidine (3,3'-DCB) on urinary bladder carcinogenesis were examined in male Wistar rats. Each chemical was administered for 4 wk in various combinations. BBN was given in the drinking water and the other carcinogens were administered in the diet. Histologic changes of bladder epithelium were classified into four types: diffuse cell growth, focal hyperplasia, papilloma, and cancer. BBN for 4 wk resulted in no histopathological changes, mild diffuse cell growth, and/or focal hyperplasia after 4, 8, 12, or 16 wk of observation. No bladder carcinomas were present in rats given only BBN or any one of the other three chemicals. Statistically significant incidences of bladder carcinomas occurred with the sequential administration of all four chemicals or the first three chemicals without 3,3'-DCB. Bladder cancer was also present in rats treated sequentially with FANFT, 2-FAA, and 3,3'-DCB. No antagonistic effects among the chemicals were observed. (30 refs.)

**77-1861 Intramembrane Particles (IMP) in Chemical Carcinogen-Induced Rat Urinary Bladder Carcinoma (Meeting Abstract).** (Eng.) Pauli, B. U. (Dept. Pathology, Rush Medical Coll., Chicago, IL 60612) Weinstein, R. S.; Alroy, J.; Friedell, G. H. *Fed Proc* 36(3): 1086; 1977. (no refs.)

**77-1862 Mutagenic Evaluation of Nitrofurans Derivatives in *Salmonella typhimurium*, by the Micronucleus Test, and by In Vivo Cytogenetics.** (Eng.) Goodman, D. R. (Dept. Pharmacology, Sch. Medicine, Univ. California, San Francisco, CA 94143) Hakkinen, P. J.; Nemenzo, J. H.; Vore, M. *Mutat Res* 48(3/4): 295-306; 1977.

The mutagenicity of 12 nitrofurans was tested in *Salmonella typhimurium* strains TA100 and TA98, and all 12 were found to be mutagenic in both strains. Nitrofurazone and nitrofurantoin dose-response curves indicated that TA100 was a more sensitive indicator of nitrofurans mutagenic activity than TA98. The mutagenicity of nitrofurazone and nitrofurantoin was also evaluated by the micronucleus tests in bone marrow cells of male Sprague-Dawley rats. The test compounds were given ip, one half the dose 30 hr, and the rest 6 hr, before sacrifice. Neither nitrofurazone at 15, 30, and 60 mg/kg nor nitrofurantoin at 50, 100, and 200 mg/kg increased the percentage of reticulocytes with micronuclei significantly. Furfuryluramide, 60, 120, and 240 mg/kg, increased the percentage of micronuclei slightly. In in vivo cytogenetic testing, single dose of 60 mg/kg nitrofurazone ip did not induce chromosomal aberrations in rat bone marrow samples after 6 and 24 hr. These findings suggest that the TA100 and TA98 strains are intrinsically more sensitive in detecting the mutagenicity of nitrofurans compounds than the in vivo mammalian tests used. The results also indicate that the mutagenic and carcinogenic properties of nitrofurans are not always positively correlated. (25 refs.)

**77-1863 Hypochlorhydria, Gastric Cancer, and Gastric Juice Nitrite Concentrations (Meeting Abstract).** (Eng.) Ruddell, W. S. (Dept. Gastroenterology, Central Middlesex Hosp., London, England) Bone, E. S.; Walters, C. L.; Blendis, L. M. *Gut* 17(10): 831-832; 1976. (2 refs.)

**77-1864 Nitrosopyrrolidine Formation in Fried Bacon.** (Eng.) Hwang, L. S. (Hoffman-La Roche, Incorporated, Nutley, NJ 07110) Rosen, J. D. *J Agric Food Chem* 24(6): 1152-1154; 1977.

The conversion of radiolabeled proline, spermidine, and putrescine to nitrosopyrrolidine (NPY) was determined after they were injected into 250-mg slices of bacon and heated under conditions analogous to those used in the normal preparation of fried bacon (185 C for 5 min). The percent conversion of proline to NPY was 0.328%, 0.27%, 0.261%, and 0.148% in bacon slices containing 200, 100, 50, and 25 ppm of sodium nitrite, respectively. No conversion of radiolabeled spermidine or putrescine could be detected. It is concluded that proline is more likely to be the precursor of NPY found in fried bacon than either spermidine or putrescine. (20 refs.)

**77-1865 Effect of Vitamin A on Formation, Toxicity and Carcinogenicity of Nitroso-N-Methylbenzylamine.** (Eng.) Schweinsberg, F.; Schott-Kollat, P. In: *Environmental N-Nitroso Compounds: Analysis and Formation. Proceedings of a Working Conference held at the Polytechnical Institute, Tallinn, Estonian SSR, 1-3 October 1975.* Walker, E. A.; Bogovski, P.; Gričiute, L.; Davis, W., eds. International Agency for Research on Cancer. (Lyon, France): IARC Scientific Publications No. 14, pp. 453-459; 1976.

An investigation was made of the effects of vitamin A on the reaction of N-methylbenzylamine (MBA) with nitrite in dilute acidic solution and of the toxicity and carcinogenicity of nitroso-N-methylbenzylamine (NMBA). The decomposition of nitrite by vitamin A in a buffered solution at pH 3.3 was observed: only 70% of 2 µg of nitrite in a 10-ml solution with 70 mg of vitamin A was recovered after 30 min at room temperature. However, the addition of 0.02 N vitamin A had no effect on the reaction of 0.01 N MBA with 0.2 N sodium nitrite, which yielded about 2.5% NMBA. The acute toxicity of NMBA in rats was found to be highly dependent upon dietary vitamin A content. After a single oral dose of 12 mg/kg NMBA, 9/9 animals fed a vitamin A-deficient diet died within 9 days; only 1/10 rats fed an enriched diet died. However, vitamin A did not significantly increase the life span of rats receiving 10 mg NMBA chronically in drinking water or protect against esophageal tumors induced by this treatment. (13 refs.)



- 77-1866 A Search for Volatile Nitrosamines in East African Spirit. (Eng.) Gough, T. A. (Lab. of Government Chemist, London, England) *Gut* 18(4): 301-302; 1977.

Alcoholic beverages from areas of high, high-moderate, and low areas of esophageal cancer in East Africa were examined for the presence of nitrosamines. There were no volatile nitrosamines present above 0.001  $\mu\text{g}/\text{ml}$  in these samples. (3 refs.)

- 77-1867 Early Surface Changes of the Urinary Bladder Epithelium of Different Animal Species Induced by N-Butyl-N-(4-hydroxybutyl)nitrosamine. (Eng.) Shirai, T. (First Dept. Pathology, Nagoya City Univ. Medical Sch., 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya 467, Japan) Murasaki, G.; Tatematsu, M.; Tsuda, M.; Fukushima, S.; Ito, N. *Gann* 68(2): 203-212; 1977.

Early surface changes of the bladder epithelium of male Wistar rats, ICR mice, golden hamsters, and Hartley guinea pigs induced by N-butyl-N-(4-hydroxybutyl)nitrosamine (BBN, 0.05% in drinking water) were examined by light microscopy and scanning electron microscopy. The normal bladder epithelium is approx two to four cells thick in all four species. It increased diffusely to five to eight cells thick in rats and mice and to four to six cells thick in hamsters after 4 wk of BBN treatment. No increase in thickness was observed in guinea pig bladder epithelium. After treatment with BBN for 8 wk, focal hyperplasia developed in the bladder epithelium of rats and mice, but not in that of hamsters and guinea pigs. At 12 wk, papillomatous growths were observed in a few rats, focal hyperplasia in the mice, diffuse cell growth in the hamsters, but no changes in the guinea pigs. Scanning electron microscopy showed that the surface of the normal bladder epithelium of each species was smooth, with a network of fine ridges on regularly arranged cells that had no microvilli or cilia. Upon BBN treatment, microvilli were seen on the bladder surface in rats, mice, and hamsters after 4 wk but not in the guinea pigs, even after 12 wk. Cells in areas of focal hyperplasia in the rats and mice appeared irregularly arranged and their surface was uneven, with numerous microvilli. Of the four species, the rat showed the most marked changes; multiple foci of papillary mucosal projections were already present in rats after 8 wk of BBN treatment. Thus, the differences in the susceptibility of the urothelium of the bladder in different animal species to BBN were reflected in their fine structure. (19 refs.)

- 77-1868 Repair In Vivo of Rat-Liver DNA Damaged by Hepatocarcinogens (Meeting Abstract). (Eng.) Den Engelse, L. (Chemical Carcinogenesis Div., Antoni van Leeuwenhoek-Huis, The Netherlands Cancer Inst., Amsterdam, Netherlands) Philippus, E. J. *Mutat Res* 46(2): 115; 1977. (2 refs.)

- 77-1869 Short-Term Carcinogen-induced Changes in Composition of Rat Liver Chromatin Protein. (Eng.) Huang, P. H. (Sch. Pathology, Univ. New South Wales, Kensington, New South Wales 2033, Australia) Stewart, B. W. *Cancer Lett* 2(6): 341-348; 1977.

The electrophoretic mobility of rat liver chromatin proteins was studied immediately after the administration of dimethylnitrosamine (DMN). The degree of electrophoretic separation of rat liver chromatin proteins was higher than that of any previous study. Changes in the composition of the rat liver chromatin protein were noted as early as 4 hr after a single injection of 10-30 mg/kg DMN. Nonhistone bands were reduced in staining intensity, and increases in all F2 histone subfractions were noted. The staining intensity of F1 and F3 histone was unchanged throughout the experiment. By 24 hr postadministration of a nonnecrotizing dose of DMN, the proportion of chromatin proteins showed a marked increase in nonhistones and a decrease in the F2 histone subfractions. Slight differences between treated and untreated samples were evident at 48 hr, suggesting that at least this much time is required for recovery from the in vivo methylation. (21 refs.)

- 77-1870 The Absorption and Metabolism in Rats of Small Oral Doses of Dimethylnitrosamine: Implication for the Possible Hazard of Dimethylnitrosamine in Human Food. (Eng.) Diaz Gomez, M. I. (Laboratorio de Quimica Biotoxicologica, Instituto de Investigaciones Cientificas y Tecnicas de las Fuerzas Armadas, Zufriategui y Varela, 1603 Villa Martelli Pcia de Buenos Aires, Argentina) Swann, P. F.; Magee, P. N. *Biochem J* 164(3): 497-500; 1977.

Groups of female Wistar rats were given one dose of the carcinogen dimethylnitrosamine by gastric intubation. Absorption of  $^{14}\text{C}$ -labeled DMN from the gastrointestinal tract was measured after a 2-mg/kg dose and alkylation of liver and kidney DNA was measured after doses of 0.1  $\mu\text{g}/\text{kg}$  to 10 mg/kg. The 2-mg/kg dose was rapidly absorbed. The methylation of liver DNA was proportional to dose, suggesting that small doses are absorbed from the gut with no more loss than large doses. As the dose was decreased, there was a disproportionately greater decrease in the alkylation of kidney DNA; when the dose was  $< 40 \mu\text{g}/\text{kg}$ , methylation of kidney DNA was not detected. This possibly explains why small amounts of DMN in the diet do not induce kidney tumors. Comparison of the relative alkylation of liver and kidney DNA resulting from po and iv doses of DMN suggests that small amounts absorbed into the portal blood from the gut are completely metabolized by the liver and do not enter the general circulation. The implications of these results for the possible hazard of DMN in human food are discussed. (29 refs.)

- 77-1871 The Accumulation of O<sup>6</sup>-Methylguanine in the Liver and Kidney DNA of Rats Treated with

**Dimethylnitrosamine for a Short or a Long Period.** (Eng.) Nicoll, J. W. (Courtauld Inst. Biochemistry, Middlesex Hosp. Medical Sch., Mortimer St., London W1P 7PN, England) Swann, P. F.; Pegg, A. E. *Chem Biol Interact* 16(3): 301-308; 1977.

A determination was made of the accumulation of 7-methylguanine and O<sup>6</sup>-methylguanine in the liver and kidney DNA of Colworth-Wistar rats receiving two dosing regimens of dimethylnitrosamine (DMN). In the liver DNA, the amount of O<sup>6</sup>-methylguanine was only approx 58% higher 4 hr after the 11 doses than it was after a single dose, whereas the amount in the kidney DNA increased to 28 times the amount after a single dose. The amount of 7-methylguanine in the kidney DNA was 11 times greater after the 11 doses than after a single dose, but it was only 3 times greater in the liver. The accumulation of methylated bases in the DNA of Group C rats receiving a series of 10 daily ip doses of 0.7 mg/kg body wt of DMN after 11 wk administration of the compound in the drinking water at 8.5 ppm was examined. The amount of O<sup>6</sup>-methylguanine in the liver DNA was only 35% greater after 10 doses than it was after a single dose, whereas the level of 7-methylguanine was 3.5 times greater. The production of O<sup>6</sup>-methylguanine and its persistence in the DNA of the target organ may be responsible for the carcinogenic action of DMN. (21 refs.)

**77-1872 Excision of O<sup>6</sup>-Methylguanine from DNA of Various Mouse Tissues Following a Single Injection of N-Methyl-N-nitrosourea.** (Eng.) Buecheler, J. Max-Planck-Institut für Hirnforschung, 5 Köln 91, W. Germany) Kleihues, P. *Chem Biol Interact* 16(3): 325-333; 1977.

The persistence of O<sup>6</sup>-methylguanine (O<sup>6</sup>-MeG) in cerebral and hepatic DNA after a single injection of <sup>3</sup>H-N-methyl-N-nitrosourea (MNU) was evaluated. In both A/J and C3HeB/FeJ mice, O<sup>6</sup>-MeG was removed at a significantly slower rate from brain DNA than from liver DNA. From days 1-7 post-injection, the decay curves of cerebral O<sup>6</sup>-MeG concentrations demonstrated a similar slope in both strains of mice. Hepatic O<sup>6</sup>-MeG concentrations were almost twice as high in A/J than in C3HeB/FeJ mice. When O<sup>6</sup>-MeG values were plotted as a fraction of 7-methylguanine concentrations, a linear increase in this ratio was noted in brain DNA between days 1 and 7, the slope being somewhat steeper in C3HeB/FeJ mice. In liver DNA, the O<sup>6</sup>/7-methylguanine ratio was significantly higher in A/J than in C3HeB/FeJ mice. The amounts of methylated purines present 7 days after the injection of <sup>3</sup>H-MNU were determined. Both the O<sup>6</sup>-MeG concentrations and the O<sup>6</sup>/7-methylguanine ratios were highest in brain and lung DNA. The lowest values were observed in liver DNA, with intermediate amounts in kidney, spleen, small intestine, and stomach DNA. In mice, tumor location does not appear to depend solely on the formation and persistence of O<sup>6</sup>-alkylguanine in DNA. (24 refs.)

**77-1873 The Action of N-Methyl-N-nitrosourea on Non-established Human Cell Lines In Vitro. I. Cell Cycle Inhibition and Aberration Induction in Diploid and Down's Fibroblasts.** (Eng.) Kaina, B. (Zentralinstitut für Genetik und Kulturpflanzenforschung, Akademie der Wissenschaften der DDR, Gatersleben 4325, E. Germany) Waller, H.; Waller, M.; Rieger, R. *Mutat Res* 43(3): 387-400; 1977.

The effect of N-methyl-N-nitrosourea (MNU: 0.5 and 1 mM) on the cell cycle, DNA synthesis, and chromosomal sensitivity of cultivated diploid fibroblasts and fibroblasts with trisomy 21 was investigated in vitro. With the exception of the inhibition of G<sub>2</sub>, Down's cells were more sensitive than diploid cells with respect to decrease of the mitotic and labeling index, inhibition of the progression of cells through the early and middle S and frequency of induced chromosomal aberrations. The chromosomal sensitivity was dependent on the position of the cells in the cell cycle during treatment with MNU. If treated during late S, no differences concerning the S block and aberration frequencies were found between diploid and Down's cells. However, if MNU treatment took place during middle and early S, Down's cells were more sensitive. The higher aberration frequencies in Down's cells resulted from elevated levels of chromatid breaks, multiple fragmentations, and chromatid translocations. The possibility that the increased sensitivity of Down's cells is due to factors involved in the induction and repair of DNA lesions is discussed. The higher frequency of tumors in patients with Down's syndrome may, therefore, be the result of alterations in DNA repair and the cytological consequences. (58 refs.)

**77-1874 The Action of N-Methyl-N-nitrosourea on Non-established Human Cell Lines In Vitro. II. Non-random Distribution of Chromatid Aberrations in Diploid and Down's Cells.** (Eng.) Kaina, B. (Zentralinstitut für Genetik und Kulturpflanzenforschung, Akademie der Wissenschaften der DDR, Gatersleben 4325, E. Germany) *Mutat Res* 43(3): 401-413; 1977.

Chromatid gaps, breaks, and aberrations involved in interchanges induced by N-methyl-N-nitrosourea (MNU:  $1 \times 10^{-3}$  M) were nonrandomly distributed on individual chromosomes and chromosome segments (G bands) both in human diploid fibroblasts and fibroblasts with trisomy 21 cultured in vitro. Aberration events were located exclusively in pale G bands. Considering cells in the first posttreatment mitosis, the pattern of aberration distribution, as revealed by the position of the hot spots, varied with recovery time and was different in diploid and Down's cells. In comparison with diploid cells, the X chromosomes of Down's cells were not involved in aberrations. Despite the higher aberration frequencies of Down's cells, the number of hot spots and the proportion of aberrations located within them were not increased in this cell type. Therefore the increased chromosomal sensitivity to MNU of Down's cells does not reflect an



increased sensitivity of special chromosomes or chromosome sites. (52 refs.)

- 77-1875 Alkylation of Cytosine and 5-Hydroxymethylcytosine by Methyl Methanesulphonate and N-Methyl-N-nitrosourea: Its Relevance to Mutagenesis.** (Eng.) Smith, B. J. (Chester Beatty Res. Inst., Inst. Cancer Res., Royal Cancer Hosp., London, England) *Chem Biol Interact* 16(3): 275-280; 1977.

The reaction of cytosine and 5-hydroxymethylcytosine (OHMeCyt) with monofunctional alkylating agents was studied to determine (1) the possible role of cytosine alkylation in mutagenesis and (2) the possibility that the immunity of T-even phages to mutation by methyl methanesulfonate (MMS) was due to the unreactivity of OHMeCyt toward this agent. Both MMS and methyl nitrosourea (MNU) reacted with both of the bases, but although ethylated and isopropylated cytosine and OHMeCyt would be expected to be separated more easily from their parent bases than the methylated bases, this did not occur. OHMeCyt and cytosine reacted equally well with MNU and MMS, affording < 1% and approx 6%, respectively, of the 3- substituted derivative. The product of cytosine methylation was identical to authentic 3-methylcytosine. The putative 5-hydroxymethyl-3-methylcytosine proved unsuitable for analysis by mass spectroscopy. After subjection of the bases to reaction with isopropyl methanesulfonate (iPMS), N-ethyl-N-nitrosourea (ENU), or ethyl methanesulfonate (EMS), no product was isolated. The results disallow the possibility that T-even phages resist mutation by MMS owing to the nonreactivity of OHMeCyt, unless it is that the reactivity is suppressed in the corresponding nucleotide. They indicate that the alkylation of OHMeCyt plays no role in the mutagenization of T-even phages by EMS, ENU, or iPMS. (24 refs.)

- 77-1876 Chromosomal Aberration, Mutation and Morphological Transformation of Syrian Hamster Embryonic Cells after Exposure to Methylnitrosocyanamide.** (Eng.) Inui, N. (Dept. Experimental Pathology, Cancer Inst., Tokyo, Toshima-ku, Tokyo 170, Japan) Taketomi, M. *Mutat Res* 43(3): 429-440; 1977.

Hamster embryo fibroblasts were treated directly for 3 hr with  $2.50 \times 10^{-6}$  M methylnitrosocyanamide (MNC), a nitrosated product of methylguanidine (MG), or with  $1.34 \times 10^{-6}$  M N-methyl-N'-nitro-N-nitrosoguanidine (MNNG). They were examined for chromosomal aberrations, morphological transformation, and mutations resistant to 8-azaguanine (8AG) and 6-thioguanine (6TG). Direct treatment with  $2.10 \times 10^{-6}$  M MNC caused a marked, dose-dependent appearance of 8AG- and 6TG-resistant mutations. The ability of MNC to induce mutations was similar to that of MNNG. Cultured embryo fibroblasts in metaphase plates

also showed a marked dose-dependent increase in chromosomal aberrations within 24 hr after direct treatment with MNC or MNNG. Moreover, MNC and MNNG caused similar rates of morphological transformation. In combination with suitable activation systems, the mutagenic assay test described should be useful for detecting nitrosation products and other hazardous substances in the environment. (31 refs.)

- 77-1877 Enhancement of DNA Polymerase II Activity in E. coli after Treatment with N-Methyl-N'-nitro-N-nitrosoguanidine.** (Eng.) Miyaki, M. (Dept. Biochemistry, Tokyo Metropolitan Inst. Medical Science, 3-18, Honkomagome, Bunkyo-ku, Tokyo 113, Japan) Sai, G.; Katagiri, S.; Akamatsu, N.; Ono, T. *Biochem Biophys Res Commun* 76(1): 136-141; 1977.

The mechanism by which N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) affects replication repair was investigated by treating *Escherichia coli* P3478 (polA1, thy-) with MNNG and then incubating the cells in growth medium for 1 hr. Immediately after treatment of bacteria with MNNG, DNA polymerase II and III activities were the same as those of controls. Incubation for 1 hr resulted in an increase of polymerase II activity up to 5 times that of control cells; polymerase III activity was unchanged or increased not more than 1.5 times. The enhancement of polymerase II activity was inhibited by chloramphenicol (CM) in the post-treatment incubation medium. During the 18-hr incubation of MNNG-treated cells with CM, the cellular damage caused by MNNG was preserved, but polymerase II activity increased when the antibiotic was removed. It is concluded that enhancement of polymerase II activity is caused via protein synthesis after MNNG treatment. Although the participation of polymerase II in DNA repair and/or mutagenesis is not yet clear, the results reflect the simultaneous induction of changes in DNA polymerase, the filamentous growth of cells, and the MNNG mutagenesis caused by the interaction of MNNG with DNA and other cellular components. (14 refs.)

- 77-1878 Effects of Nutrition and Microflora on Hepatic and Intestinal Mixed Function Oxidases (Meeting Abstract).** (Eng.) Martin, C. W. (Naylor Dana Inst., Valhalla, NY 10595) Reddy, B. S.; Weisburger, J. H. *Fed Proc* 36(3): 1148; 1977. (no refs.)

- 77-1879 Malignant Transformation of Pancreatic Ductal Epithelium In Vitro with N-Methyl-N-Nitrosoguanidine (MNNG) (Meeting Abstract).** (Eng.) Jones, R. T. (Univ. Maryland Sch. Medicine, Dept. Pathology, Baltimore, MD 21201) Resau, J.; Bostwick, D.; Trump, B. F. *Fed Proc* 36(3): 1079; 1977. (no refs.)

77-1880 **4-Hydroxyaminoquinoline-1-oxide-Induced Ultrastructural Changes in the Guinea-Pig Exocrine Pancreas.** (Eng.) Rao, M. S. (Dept. Pathology and Oncology, Univ. Kansas Medical Center, Coll. Health Sciences and Hosp., Kansas City, KS 66103) *J Pathol* 120(2): 109-114; 1976.

Ultrastructural changes induced by 4-hydroxyaminoquinoline-1-oxide (HAQO) in the guinea pig exocrine pancreas were studied. Between 18 and 24 hr after a single dose of HAQO (22.5 mg/kg, iv), the nucleoli of the acinar cells appeared somewhat larger than normal and the fibrillar and granular components were segregated into distinct areas. Nucleoplasmic interchromatin granules were prominent. Cytoplasmic alterations were characterized by minimal dilatation of the rough endoplasmic reticulum throughout the cell. The membrane attached and the free ribosomes appeared unaltered. The intracisternal granules were decreased significantly. The mitochondria were not altered markedly, but an occasional one was swollen, with disruption or focal dilatation of the outer membrane. The mature zymogen granules were significantly decreased, and the apical cytoplasm was occupied by an increased number of immature granules. There was a moderate increase in the number of cytoplasmic dense bodies in acinar cells. By light microscopy the peak degenerative changes in the exocrine pancreas were observed at 55 hr, although few scattered acini or several cells in a particular acinus appeared unaffected. By electron microscopy, the rough endoplasmic reticulum demonstrated vesicular to cisternal dilatation containing either electron-lucent or finely fibrillar electron-dense material. The focal mitochondrial changes progressed, with disruption of the limiting membranes. The zymogen granules were markedly decreased or completely absent in most cells. The size of the Golgi complex was reduced markedly. The hyaloplasm contained pleomorphic cytoplasmic degenerative bodies composed of membrane-bound vacuoles and/or myelin figures. At 43 hr, some acinar cells displayed lipid droplets in the cytoplasm and the nucleus. By 55 hr, numerous acinar cells were necrotic. Between 43 and 60 hr, the nucleoli were small and appeared condensed or fragmented. Both centroacinar and ductal cells showed degenerative changes, including organelle changes, cytoplasmic degenerative bodies, increased lysosomes, and fat vacuoles. The acute effects of HAQO on the pancreas were mainly necrotic. (33 refs.)

77-1881 **The Lesions That Lead to Sister Chromatid Exchanges Are Different from Those That Lead to Chromosome Aberrations (Meeting Abstract).** (Eng.) Wolff, S. (Lab. Radiobiology and Dept. Anatomy, Univ. California, San Francisco, CA 94143) *Mutat Res* 46(2): 164; 1977. (no refs.)

77-1882 **Evaluation of the Effect of Ethanol on the Frequency of Micronuclei in the Bone Marrow of Swiss Mice.** (Eng.) Chaubey, R. C. (Bhabha Atomic Res.

Centre, Bio-Medical Group, Trombay, Bombay 400 085, India) Kavi, B. R.; Chauhan, P. S.; Sundaram, K. *Mutat Res* 43(3): 441-444; 1977.

Ethanol failed to increase the frequency of micronuclei in polychromatic and monochromatic RBC in the bone marrow of male Swiss mice, nor were dominant-lethal mutations increased after 5 to 6 wk ingestion of alcohol. (14 refs.)

77-1883 **L-Ethionine Acts to Induce Globin Synthesis in Friend Erythroleukemia Cells (Meeting Abstract).** (Eng.) Christman, J. (Dept. Biochemistry, Mount Sinai Sch. Medicine CUNY, New York, NY 10029) Price, P.; Charrow, J.; Pedrinan, L.; Acs, G. *Fed Proc* 36(3): 819; 1977. (no refs.)

77-1884 **Sequential Alterations in the Hepatic Content and Metabolism of Cyclic AMP and Cyclic GMP Induced by DL-Ethionine: Evidence for Malignant Transformation of Liver with a Sustained Increase in Cyclic AMP.** (Eng.) DeRubertis, F. R. (Veterans Admin. Hosp., Univ. Drive C, Pittsburgh, PA 15240) *Metabolism* 25(12): 1611-1625; 1976.

The sequential changes in the hepatic content and metabolism of cyclic guanosine monophosphate (GMP) and cyclic AMP induced by DL-ethionine were studied. Both the protein and DNA content of livers from rats fed ethionine for 2 wk were less than control levels. The cyclic AMP of livers from ethionine-treated rats was significantly higher than that of control livers at 2 wk. This difference persisted and was apparent when cyclic AMP content was expressed on the basis of tissue protein or DNA content as well as wet wt. The cyclic GMP content in surrounding tissue from tumor-bearing rats did not differ from that of control liver, whether expressed on the basis of wet wt, protein, or DNA. However, in hepatomas from ethionine-fed rats, both cyclic AMP and cyclic GMP were increased relative to values in either the surrounding liver from the same rats or to those of control liver. The ratio of cyclic AMP to cyclic GMP content was decreased in the tumors (15.4) compared to the surrounding liver (26.3), but not relative to that of the control liver (13.5). The basal adenylate cyclase activity of livers from ethionine-fed rats was significantly higher than that of controls. This difference was detectable by 2 wk and persisted throughout the study. Cyclic AMP-independent protein kinase activity was significantly higher in whole homogenates of uninvolved liver from ethionine-fed rats compared to corresponding control values. The proportion of total physiologically active glycogen synthetase was clearly reduced in tissues from ethionine-fed rats. The cyclic AMP content of surrounding liver from tumor-bearing rats (2,261 picomoles/g wet wt) remained significantly higher than that of liver from pair-fed controls (1,058) following in vitro incubation of slices of these



tissues. Despite the higher basal cyclic AMP levels in tissues from the ethionine-treated rats, both absolute and relative cyclic AMP responses to a max stimulating concentration of glucagon (10  $\mu$ M) were blunted but not absent. The results indicate that malignant conversion can occur in the liver with a sustained elevation of both total and effective cyclic AMP during the premalignant phase. (66 refs.)

**77-1885 Physicochemical Changes in DNA Exposed to Alkylating Carcinogens and Mutagens (Meeting Abstract).** (Eng.) Kubinski, H. (Univ. Wisconsin, Madison, WI 53706) Kubinski, Z. O.; Morin, N. R. *Fed Proc* 36(3): 305; 1977. (no refs.)

**77-1886 Mutagenic Effect of Epichlorohydrin. II. Analysis of Chromosomal Aberrations in Lymphocytes of Persons Occupationally Exposed to Epichlorohydrin.** (Eng.) Kucerova, M. (Genetic Lab. Inst. Hygiene and Epidemiology, Prague, Czechoslovakia) Zhurkov, V. S.; Polivkova, Z.; Ivanova, J. E. *Mutat Res* 48(3/4): 355-360; 1977.

Peripheral lymphocytes from 35 workers (23-54 yr old) occupationally exposed to epichlorohydrin (ECHH) were analyzed cytogenetically before and 1 and 2 yr after exposure. The workers, who were generally healthy and had not been irradiated or treated with other known mutagens, were exposed to 0.5-5.0 mg ECHH/m<sup>3</sup>. The lymphocytes were cultivated for 56-58 hr before conventional Giemsa staining. Four slides were prepared from each donor, and two were scored independently in each of two collaborating laboratories. The amount of cells with chromosomal aberrations was 1.37% before exposure, 1.91% after the first yr, and 2.69% after the second yr. Detected aberrations fell into four groups: (1) chromatid breaks, (2) chromatid exchanges, (3) chromosome breaks, and (4) chromosomal exchanges. The increased number of aberrant cells was mainly caused by chromatid and chromosomal breaks, but chromatid and chromosomal exchanges appeared to be rare in all cells sampled. These results confirm the assumption that ECHH, at dose levels exceeding the max acceptable concentration, has a mutagenic effect on chromosomes in the peripheral lymphocytes of exposed persons. (16 refs.)

**77-1887 Alkylation of Nucleic Acids by Metabolites of Vinyl Chloride In Vitro and In Vivo: Formation of 1-N<sup>6</sup>-Etheno-Adenosine (Meeting Abstract).** (Eng.) Laib, R. J. (Inst. fur Toxikologie Univ. Tubingen, Wilhelmstr. 56, D-7400 Tubingen-1, W. Germany) Bolt, H. M. *Archiv fur Pharmakologie (Berlin)* 297(11, Suppl): R22; 1977. (no refs.)

**77-1888 Vinyl Chloride Mutagenesis in *Drosophila melanogaster*.** (Eng.) Verburgt, F. G. (Dept. Radiation Genetics and Chemical Mutagenesis, State Univ. Leiden, Sylvius Labs., Leiden, Netherlands) Vogel, E. *Mutat Res* 48(3/4): 327-336; 1977.

The mutagenicity of vinyl chloride monomer (VCM) was tested in *Drosophila melanogaster* males at low and high concentrations by comparing short-term vs long-term exposure and by using different genetic end points. In inhalation experiments, groups of 50 *Drosophila* males were exposed to VCM at concentrations of 30, 200, 850, 10,000, 30,000 or 50,000 ppm for 2 days and to 30 or 850 ppm for 17 days. Exposed males were mated, and the mutagenic response to VCM during successive stages of spermatogenesis was tested by brood pattern analysis. All suspected recessive lethals were verified by retests. VCM was mutagenic in the recessive-lethal test after both short- and long-term exposures. The lowest effective concentration was 850 ppm after 2 days exposure and 30 ppm after 17 days. The mutation frequency increased with concentration and reached a plateau at 10,000 ppm, which indicates a substrate saturation effect. Negative results were obtained when tests on dominant lethals, translocations, and entire and partial sex-chromosome loss were carried out with VCM at 30,000 ppm for 2 days. These results document the high resolving power of the *Drosophila* recessive-lethal test and support the view that chromosome breakage is not a reliable measure of mutagenic activity. (25 refs.)

**77-1889 Vinyl Chloride-Mediated Cytochrome P-450 Destruction (Meeting Abstract).** (Eng.) Strickland, T. W. (Vanderbilt Univ., Nashville, TN 37232) Guengerich, F. P. *Fed Proc* 36(3): 991; 1977. (no refs.)

**77-1890 Mutagenicity of Chlorinated Cyclopentadienes Due to Metabolic Activation (Meeting Abstract).** (Eng.) Bonse, G. (Dept. Toxicology Univ. Wurzburg, D 87 Wurzburg, Versbacher Landstrasse 9, Wurzburg, W. Germany) Goggelmann, W. *Archiv fur Pharmakologie (Berlin)* 297(11, Suppl): R22; 1977. (no refs.)

**77-1891 Health Aspects of the Curing of Synthetic Rubbers.** (Eng.) Fraser, D. A. (Dept. Environmental Sciences, Univ. North Carolina, Chapel Hill, NC 27514) Rappaport, S. *Environ Health Perspect* 17: 45-53; 1976.

Volatile substances emitted during the curing process of a commonly used tread rubber formulation were analyzed by combined gas chromatography-mass spectrometry. Compounds identified included styrene, butadiene oligomers, alkylbenzenes and naphthalenes, and several specific nitrogen- and sulfur-containing substances. The concentrations of these

compounds were so low that acute toxicity problems appear unlikely; but since little is known about their chronic toxicities, the possibility of significant health effects cannot be ruled out. (47 refs.)

- 77-1892 **An Extraction Method for Determination of Benzene in Tissue by Gas Chromatography.** (Eng.) Snyder, C. A. (New York Univ. Medical Center, Inst. Environmental Medicine, 550 First Ave., New York, NY 10016) Erlichman, M. N.; Goldstein, B. D.; Laskin, S. *Am Ind Hyg Assoc J* 38(6): 272-276; 1977.

An extraction/gas chromatography procedure was used to determine benzene levels in the liver, spleen, bone marrow, and blood of mice and the bone marrow of rats after exposure to benzene vapor. The distribution of benzene was assessed in the tissues of 9-mo-old male AKR mice receiving a single 6-hr exposure to benzene vapor. In experiments to determine extraction efficiencies, several samples were spiked with the following amounts of benzene (per unit of material): liver, 2.81-5.21  $\mu\text{g/g}$ ; spleen, 3.94-6.00  $\mu\text{g/g}$ ; rat bone marrow, 15.8-35.7  $\mu\text{g/g}$ ; mouse bone marrow, 8.5-24.0  $\mu\text{g/g}$ ; and blood, 8.80  $\mu\text{g/ml}$ . The recovery efficiencies were liver, 47.4%; spleen 48.4%; rat bone marrow, 35.4%; mouse bone marrow, 38.05; and blood, 60.8%. Unmetabolized benzene was detected in the bone marrows of AKR mice following a single exposure to 300 or 100 ppm benzene vapors. The results were reproducible even with very small quantities of sample (0.05 g for tissue and 0.02 ml for blood). The procedure allows for reproducible analysis of benzene in small tissue samples following exposure to levels similar to those found in industrial environments. (9 refs.)

- 77-1893 **Report on Carcinogenesis Bioassay of Technical Grade Chlordecone (Kepone).** (Eng.) Anonymous (Carcinogenesis Program, NCI, Bethesda, MD 20014) *Am Ind Hyg Assoc* 37(12): 680-681; 1977.

Technical-grade chlordecone [Kepone: decachlorooctahydro-1,3,4-metheno-2H-cyclobuta(cd)pentalen-2-one] was tested for carcinogenicity in B6C3F1 mice. Of 49 male mice fed a diet containing 23 ppm Kepone for 80 wk, 43 developed liver cancers upon sacrifice after a further 10 wk. Liver cancers also developed in 39/48 male mice fed 20 ppm Kepone, in 23/49 female mice fed 40 ppm, and in 26/50 female mice fed 20 ppm. Untreated female mice did not develop any liver tumors; a low spontaneous incidence of these tumors was seen in male mice. Of 44 male Osborne-Mendel rats fed 24 ppm Kepone for 80 wk, 3 developed liver cancer after a further 32 wk; 10/45 female rats fed 26 ppm Kepone developed liver tumors. No liver cancer was seen in control rats of either sex. Extensive liver hyperplasia was seen in all treated rodents. It is concluded that Kepone is hepatocarcinogenic in rodents. (no refs.)

- 77-1894 **Carcinogenicity of DDT (Dichlorodiphenyl Trichloroethane) in Pure Inbred Swiss Mice.** (Eng.) Kashyap, S. K. (Nat'l. Inst. Occupational Health, Ahmedabad-380016, India) Nigam, S. K.; Karnik, A. B.; Gupta, R. C.; Chatterjee, S. K. *Int J Cancer* 19(5): 725-729; 1977.

Inbred Swiss mice were treated with technical-grade 2,2-bis(p-chlorophenyl)-1,1,1-trichloroethane (DDT) (1) po with the diet (100 ppm) or by intubation (0.25 mg), (2) sc (0.25 mg), and (3) by skin painting (0.25 mg in 0.1 ml olive oil). The total duration of the experiment was 80 wk. There was no difference in body growth and mortality between the experimental and control groups. The toxic manifestations of DDT were tremors, convulsions, and corneal opacity usually after 40 wk. Po and sc DDT treatment resulted in a significant increase in the incidence of tumors, mainly of the lymphoid tissues, lung, and liver. The highest tumor incidence was recorded in the group of mice receiving DDT sc. Males and females were equally susceptible. No evidence of carcinogenicity was observed in the painted group. (12 refs.)

- 77-1895 **Lack of Carcinogenicity of DDT in Hamsters (Meeting Abstract).** (Eng.) Cabral, J. R. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NB 68105) Shubik, P. *Fed Proc* 36(3): 1086; 1977. (no refs.)

- 77-1896 **The Metabolism of Cyclohexanecarboxylate in the Rat.** (Eng.) Brewster, D. (Dept. Drug Metabolism, Pharmaceutical Div., Reckitt and Colman, Dansom Lane, Hull HU8 7DS, England) Jones, R. S.; Parke, D. V. *Biochem J* 164(3): 595-600; 1977.

$^{14}\text{C}$ -cyclohexanecarboxylate (CHC: 5-6  $\mu\text{gCi}$ , 0.5-200 mg/kg) was administered to male Wistar rats as the sodium salt in 0.5 ml water via the duodenal canula. Analysis of ethyl acetate extracts of urine and bile by electron-impact mass spectrometry and paramagnetic resonance spectrometry showed that CHC was metabolized and excreted (mostly in the urine) as hippurate, hexahydrohippurate, 3,4,5,6-tetrahydrohippurate, and the benzoyl and cyclohexylcarbonyl  $\beta$ -glucuronides. The pattern of metabolism was dose-dependent. With decreasing dose, a progressive increase in the conversion into hippurate occurred. This was largely at the expense of glucuronide formation, although the proportions of hexahydro- and tetrahydrohippurate were also decreased. The observed formation of hexahydrohippurate and 3,4,5,6-tetrahydrohippurate substantiates the proposed mechanism of aromatization of CHC. It appears that these compounds arise via glycine conjugation of active intermediates during aromatization. Hexahydrohippurate and 3,4,5,6-tetrahydrohippurate may occur in the urine of rats as new metabolites of shikimate, dependent for their formation on microbial metabolism. (21 refs.)



- 77-1897 **Effects of Carmine and Carminic Acid on Embryonic Tissue Cell Cultures.** (Eng.) Marzona, L. (Anatomical Inst., Medical Faculty Univ. Modena, via Berengario 16, I-41100 Modena, Italy) Olivo, O. M.; Volpi, G.; Toni, G. *Experientia* 33(6): 755-756; 1977.

Varying doses of carmine and carminic acid did not produce harmful effects on the metabolism and proliferation of cultured chicken and mammalian embryonic tissue cells. (2 refs.)

- 77-1898 **The Penetration of N-Hydroxyurethane into the Prostate and Prostatic Secretion of the Rat and Dog.** (Eng.) Smith, E. R. (Dept. Pharmacology, Univ. Massachusetts Medical Sch., Worcester, MA 01605) Hagopian, M.; Norlin, R. D. *Toxicol Appl Pharmacol* 40(2): 335-345; 1977.

The uptake and secretion of N-hydroxyurethane (HU) by the rat and dog prostate were studied. In unanesthetized rats followed for up to 6 hr after the iv administration of 100 mg/kg of HU, the compound was readily detectable in the plasma and prostatic fluid. Detectable quantities of HU were found in the ventral and dorsolateral prostates only 2 min after treatment. Over the first 2 hr after treatment, the mean gland-to-plasma ratios were 1.33 for the ventral prostate and 0.91 for the dorsolateral prostate. Six dogs, four with prostatic fistulas, were given iv injections of 100 mg/kg of HU. Plasma concentrations were highest immediately after administration and then fell rapidly. HU could not be detected in the plasma several hours after treatment. HU was found in the prostatic fluid 1 hr after treatment, but it could not be detected in samples taken thereafter. The mean gland-to-plasma ratio for four dogs examined at 120 min after treatment was 2.60. The results indicate that HU penetrates the prostate gland and its secretions in both the rat and the dog. (10 refs.)

- 77-1899 **Tumorigenesis with 4-Methylphenylhydrazine Hydrochloride in Mice (Meeting Abstract).** (Eng.) Toth, B. (Eppley Inst. Res. Cancer, Univ. Nebraska, Omaha, NB 68105) *Fed Proc* 36(3): 1087; 1977. (no refs.)

- 77-1900 **The Enhancing Effect of Sodium Barbitol on Metastatic Prostate Adenocarcinomas (CAS) in Rats (Meeting Abstract).** (Eng.) Pollard, M. (Lobund, Notre Dame, IN 46556) Luckert, P. H. *Fed Proc* 36(3): 1087; 1977. (no refs.)

- 77-1901 **In Vitro Differentiation of Early Mouse Embryos After Cyclophosphamide Treatment (Meeting Abstract).** (Eng.) Eibs, H. G. (Dept. Toxicology

and Prenatal Pharmacology, Garystr. 1-9, D-1 Berlin 33, W. Germany) Jacob, U. *Archiv fur Pharmakologie (Berlin)* 297(11, Suppl): R9; 1977. (no refs.)

- 77-1902 **Some Effects of Diet on Cyclamate Toxicity (Meeting Abstract).** (Eng.) Pritchard, A. B. (Cornell Univ., Ithaca, NY 14853) Warner, R. G. *Fed Proc* 36(3): 1117; 1977. (no refs.)

- 77-1903 **Chromosome Studies of Bone Marrow Cells and Peripheral Blood Lymphocytes from Diphenylhydantoin-treated Patients.** (Eng.) Alving, J. (Section Hematology, Dept. Medicine B, Blood Bank and Blood grouping Lab., Aalborg Hosp., Aalborg, Denmark) Jensen, M. K.; Meyer, H. *Mutat Res* 361-366; 1977.

Chromosome studies were performed on the bone marrow cells and peripheral blood WBC from 10 patients with epilepsy (4 women, 6 men, 19-49 yr old) who had been treated with diphenylhydantoin alone for 4-20 yr. There was no significant increase either in metaphases with structural abnormalities or in hyperdiploid metaphases in the treated patients compared to controls. Bone-marrow smears from five of the patients were studied for the presence of micronuclei. There was no increase in the number of micronuclei compared with controls. Although animal experiments have shown a distinct teratogenic effect of diphenylhydantoin and most of the congenital defects in children born of epileptic mothers are cleft palate and/or lip, there is at present no convincing evidence that diphenylhydantoin per se is teratogenic in man. Cytogenetic investigations with chemical agents should be performed on tissues that are exposed to the drug in vivo and those that do not need to be cultured in vitro before preparation of chromosomes; ie, bone marrow cells and/or germ cells. (22 refs.)

- 77-1904 **Wilms Tumor in an Adult Associated with Androgen Abuse.** (Eng.) Prat, J. (Dept. Pathology, New York Hosp.-Cornell Medical Center, 525 E. 68th St., New York, NY 10021) Gray, G. F.; Stolley, P. D.; Coleman, J. W. *JAMA* 237(21): 2322-2323; 1977.

A 38-yr-old man who had used large amounts of anabolic androgenic steroids as part of a body-building program developed Wilms' tumor. Sonography revealed a mass in the left kidney. Venacavagram showed a filling defect extending from the left kidney and propagating toward the heart. Anaplastic neoplasm was discovered in the lungs. The patient died 5 mo after surgery. (3 refs.)

- 77-1905 **Clomid or Nafoxidine Administered to Neonatal Rats Causes Reproductive Tract Abnormalities.** (Eng.) Clark, J. H. (Dept. Cell Biology, Baylor Coll. Medicine, Houston, TX 77030) McCormack, S. *Science* 197(4299): 164-165; 1977.

Reproductive tract abnormalities were investigated in neonatal Sprague-Dawley female rats injected on day 1 of life with Nafoxidine (100 µg) or Clomid (10-500 µg). Histologic analysis of ovarian and uterine tissue from rats killed at age 60-100 days revealed multiple abnormalities; these included cystic ovaries, ovarian hypoplasia, hilus cell tumors, oviductal hyperplasia, pyometra, epithelial metaplasia, uterine cystic hyperplasia, and uterine tumors. The types and frequency of abnormalities varied widely among the various treatment groups. The highest dose of either estrogen produced some form of abnormality in 80%-100% of the animals, while 10%-50% were adversely affected by intermediate and lower doses. Clomid and Nafoxidine also induced effects that resembled those found in masculinized female rats: the vaginal smears of treated rats did not show normal cyclic changes, and there was a high incidence of estrus smears. The possibility that the reproductive tract abnormalities were due to a sustained estrogenic stimulation of uterine growth is discussed. (7 refs.)

- 77-1906 **The Effect of Subcutaneous Administration of Oestrogens to Female Rats on Levels of Plasma Oestrogen and Tumour Incidence (Meeting Abstract).** (Eng.) Blankenstein, M. A. (Dept. Biochemistry, Medical Faculty, Erasmus Univ., Rotterdam, The Netherlands) van der Molen, H. J.; Broerse, J. J.; Knaan, S. *Int J Radiat Biol* 31(4): 378; 1977. (no refs.)

- 77-1907 **Metabolic Fates of Diethylstilbestrol Sulphates in the Rat.** (Eng.) Barford, P. A. (Dept. Chemistry, Univ. Aston in Birmingham, Gosta Green, Birmingham B4 7ET, England) Olavesen, A. H.; Curtis, C. G.; Powell, G. M. *Biochem J* 164(2): 423-430; 1977.

The catalysis and modes of excretion of the mono- and disulfate ester of diethylstilbestrol (DES) were studied in the rat. In one series of experiments, <sup>35</sup>S-labeled mono- or disulfate DES was administered ip (1 mg/200 g body wt) to rats in metabolism cages, and urine and feces were collected 6, 12, 24, and 48 hr later. Most of the radioactivity was found in the urine as inorganic sulfate. Mono- or disulfate DES was administered to separate sets of animals iv (1, 5, and 10 mg/100 g body wt), and the ureter and bile ducts were cannulated. After 6 hr, 46.9% of the radioactivity in males and 49.4% of the radioactivity in females showed up in the bile as an ester sulfate. Whole-body autoradiograms of the animals after DES administration demonstrated rapid accumulation in the liver; rats administered the monosulfate compound also had large amounts in the gastrointestinal tract. It was con-

cluded that both compounds undergo appreciable desulfonation and that biliary excretion is greater for the monosulfate. Autoradiograms implicate the liver as the major metabolic site of both esters. (20 refs.)

- 77-1908 **A Clinicopathologic Study of Steroid-related Liver Tumors.** (Eng.) Christopherson, W. M. (Dept. Pathology, Univ. Louisville Sch. Medicine, Health Sciences Center, Louisville, KY 40201) Mays, E. T.; Barrows, G. *Am J Surg Pathol* 1(1): 31-41; 1977.

A registry of liver tumors was started in late 1973 in an attempt to assess the relationship of these tumors to oral contraceptives or to other environmental factors. The first 101 tumors accessioned included 44 cases of focal nodular hyperplasia, 40 adenomas, 4 unclassified but probably benign tumors, and 13 hepatocellular carcinomas. Eighty-one patients took oral contraceptives, 6 were associated with pregnancy, 3 had taken estrogens for long periods of time, 1 had a thecoma, 4 never took sex steroids, and in 5 the history was unknown. Tumor rupture and intrahepatic hemorrhage were frequent complications. It is possible that the vascular lesions associated with focal nodular hyperplasia could play a part in their pathogenesis as well as the occurrence of rupture. Foci of adenomatous hyperplasia may be related to the development of adenomas. The association of these tumors with sex steroids could be coincidental. The fact that none of the patients had cirrhosis and that androgenic anabolic steroid therapy has been associated with hepatocellular carcinomas in men suggests a causal relationship (21 refs.)

- 77-1909 **Hormonal Influences on RNA-Polymerases During Carcinogenesis (Meeting Abstract).** (Eng.) Chedid, A. (Dept. Pathology, Chicago Medical Sch., Chicago, IL 60612) Bundeally, A. *Fed Proc* 36(3): 1078; 1977. (no refs.)

- 77-1910 **Inhibition of Growth and Aflatoxin Production by Cinnamon and Clove Oils, Cinnamic Aldehyde and Eugenol.** (Eng.) Bullerman, L. B. (Dept. Food Science and Technology, Univ. Nebraska-Lincoln, Lincoln, NB 68583) Lieu, F. Y.; Seier, S. A. *J Food Sci* 42(4): 1107-1109, 1116; 1977.

The effects of cinnamon oil, clove oil, cinnamic aldehyde, and eugenol on growth and aflatoxin production by *Aspergillus parasiticus* were studied using yeast extract sucrose broth as the substrate. All four substances inhibited mold growth and subsequent toxin production; cinnamon and clove oil were inhibitory at 200-2500 ppm, cinnamic aldehyde at 150 ppm, and eugenol at 125 ppm. The two latter compounds are the major products of cinnamon and clove oils and are apparent-



ly responsible for the fungistatic activity of the oils. Further study showed that the effect of cinnamic aldehyde and eugenol was inhibition of growth rather than of toxin production. Given sufficient time, cultures were inhibited initially but subsequently produced toxin levels comparable to control cultures. However, at levels above 250 ppm of oils and above 200 ppm of cinnamic aldehyde and eugenol, mold growth was either completely inhibited or so minimal that aflatoxins were not produced. (15 refs.)

- 77-1911 **Metabolic Activation of a Natural Promutagen, Eugenol, by Replicative Cultures of Adult Rat Liver Epithelial Cells.** (Fre.) Delaforge, M. (Groupe de Recherche sur la Differentiation biochimique et la Canцерогенese de Cellules eucaryotes en Culture, ERA-CNRS 267, Faculte de Medecine, 7, bd Jeanne d'Arc, 21033 Dijon, France) Janiaud, P.; Dorange, J. L.; Morizot, J. P.; Padiou, P. *C R Soc Biol* 171(1): 100-107; 1977.

The metabolism of eugenol was studied in replicative cultures of liver epithelial cells of adult male and female Wistar rats, and the mutagenic effects of eugenol and one of its metabolites, 2',3'-epoxyeugenol, were investigated in *Salmonella typhimurium*. Eugenol caused no mutations in *S. typhimurium* but 2',3'-epoxyeugenol caused point mutation exclusively, regardless of whether or not microsomes were present. (16 refs.)

- 77-1912 **Inhibition of Urease by the Mycotoxin Patulin.** (Eng.) Reiss, J. (Mikrobiologisches Laboratorium der Grahamhaus Studt K. G., Bad Kreuznach, W. Germany) *Naturwissenschaften* 64(2): 97; 1977.

The influence of the mycotoxin patulin on urease activity was studied to clarify the mechanism of patulin toxicity. A modification of the agar-diffusion technique for the determination of residues of metalliferous fungicides was used. The plates were developed with a 1% aqueous solution of urea, and the diameters of the zones remaining yellow were recorded. Higher concentrations of patulin (to 100 µg/disk) inhibited the urease activity. The detection limit was 0.2 µg/disk. (8 refs.)

- 77-1913 **Octopine and Nopaline Metabolism in *Agrobacterium tumefaciens* and Crown Gall Tumor Cells: Role of Plasmid Genes.** (Eng.) Montoya, A. L. (Dept. Microbiology and Immunology, Univ. Washington Sch. Medicine, Seattle, WA 98195) Chilton, M. D.; Gordon, M. P.; Sciaky, D.; Nester, E. W. *J Bacteriol* 129(1): 101-107; 1977.

The problem of how virulent strains of *Agrobacterium*

*tumefaciens* determine what unusual guanido amino acid will be produced in crown gall tumors was investigated. Tumors were induced on immature leaves of young kalanchoe plants by streaking octopine- or nopaline-utilizing bacterial colonies down the leaf. After 2-4 wk, the tumors were excised and analyzed for octopine/nopaline production. For a large number of *A. tumefaciens* strains, octopine-utilizing bacteria incited octopine-producing tumors and nopaline-utilizing bacteria incited nopaline-producing tumors. This indicates that octopine- and nopaline-utilization traits were carried by the same large plasmid that confers virulence. Two classes of bacteria were exceptions to this rule. One class used both octopine and nopaline, but the tumors produced only nopaline. Another class utilized nopaline, but their tumors synthesized neither nopaline nor octopine. Some strains, which were mutant in the gene specifying octopine or nopaline oxidase, still retained the permease for these amino acids as well as virulence. The tumors induced by these mutants still synthesized the same levels of octopine and nopaline as the tumors induced by their parents. These results suggest that the plasmid gene that determined octopine or nopaline production by the tumor is distinct from the plasmid gene that determines their catabolism by bacteria. (21 refs.)

- 77-1914 **Inhibition by Neonatal Hypothalamic Estrogen Implantation of Carcinogen-induced Mammary Tumorigenesis in Female Rats.** (Eng.) Hayashi, S. (Nat. Cancer Center Res. Inst., Tsukiji 5-1-1, Chuo-ku, Tokyo 104 Japan) Nagasawa, H. *Gann* 68(2): 139-143; 1977.

The relationship between neonatal treatment with estrogen and the induction of mammary tumors by 7,12-dimethylbenz(a)anthracene (DMBA) in female Sprague Dawley rats was examined. Five-day-old female rats received an intrahypothalamic implantation of micropellets of 0.4 µg of an estradiol-paraffin mixture, paraffin vehicle only (sham-operated controls), or no operation (intact controls). Induction of mammary tumors in these rats was examined after the intragastric administration of DMBA (20 mg in 1.0 ml of olive oil) at age 45 days. Rats bearing the micropellets of the estradiol-paraffin mixture in the anterior to middle part of the hypothalamus developed persistent vaginal estrus, but rats bearing the same pellets in the middle to posterior part of the hypothalamus showed regular cyclic changes in their daily vaginal smears. Compared with the intact or sham-operated controls, mammary tumor appearance was delayed in the rats that received estradiol, and they showed persistent vaginal estrus (10 wk vs 15 wk after DMBA administration respectively). Since (1) the vagina of the rats with the estradiol-paraffin mixture opened at the same age as the intact or sham-operated controls and (2) the mammary rating of the former rats was the same as the latter, it is concluded that the steroid alters the neonatal hypothalamus but has little effect on the neonatal mammary glands. Thus, the delay or suppression of mammary tumorigenesis in DMBA-treated rats by neonatal steroid administration is primarily attributable

ble to the irreversible changes in the hypothalamo-pituitary-gonadal axis. (10 refs.)

- 77-1915 **Metabolic Activation of the Carcinogen 7,12-Dimethylbenz(a)anthracene for DNA Binding.** (Eng.) Moschel, R. C. (Chemical Carcinogenesis Program, NCI Frederick Cancer Res. Center, Frederick, MD 21701) Baird, W. M.; Dipple, A. *Biochem Biophys Res Commun* 76(4): 1092-1098; 1977.

The fluorescence excitation and emission spectra of the products formed between 7,12-dimethylbenz(a)anthracene (DMBA) and the DNA of Swiss mouse embryo cell cultures were reported. Primary cultures of mouse embryo cells were grown in Eagle's Minimal Essential Medium supplemented with 10% fetal bovine serum either in plastic flasks or in roller bottles. The isolation of hydrocarbon-deoxyribonucleoside products from the DNA of cells exposed to DMBA allowed both fluorescence excitation and emission spectra to be recorded. Comparison of these spectra with those of various model compounds indicated that DMBA was metabolically activated for DNA binding through the generation of a diol-oxide in the 1, 2, 3, 4-ring. Earlier fluorescence studies have dealt only with the emission spectra of the modified DNA itself, but the present studies extend the usefulness of this approach by demonstrating that excitation spectra can also be obtained. (16 refs.)

- 77-1916 **Characterization of a Photoproduct of 7,12-Dimethylbenz(a)anthracene and Its Effects on Chick-Embryo Cells in Culture.** (Eng.) Warshawsky, D. (Lab. Chemical Biodynamics, Lawrence Berkeley Lab., Univ. California, Berkeley, CA 94720) Kerns, E.; Bissell, M. J.; Calvin, M. *Biochem J* 164(3): 481-486; 1977.

A potent impurity present in commercial 7, 12-dimethylbenz(a)anthracene (DMBA) preparations was isolated by alumina column chromatography and characterized by thin-layer chromatography, mass spectroscopy, carbon-hydrogen analysis, UV and nuclear magnetic resonance spectroscopy, and thermal decomposition. It was found to be the DMBA photooxidation product 7,12-epidioxy-7,12-dimethylbenz(a)anthracene (EDMBA). The impurity was more effective than DMBA in inducing morphological alterations and in causing an increase in glucose uptake, DNA synthesis and cell number in chick-embryo fibroblasts. Gradual morphological transformation followed the increase in DNA synthesis after 2 days when either primary or secondary cultures were treated with 3 µg/ml of EDMBA. It is suggested that some of the biological effects observed after treatment of cultures with DMBA may be due in part to the presence of EDMBA. (38 refs.)

- 77-1917 **The Epidermal Melanocytes of the Mongolian Gerbil: Their Postnatal Development and Response to Carcinogens.** (Eng.) Quevedo, W. C. (Div. Biological and Medical Sciences, Brown Univ., Providence, RI 02912) McDonald, C. J.; Dyckman, J.; Bienieki, T. C.; Fleischmann, R. D.; Holstein, T. J. *Pigm Cell* 3: 357-366; 1976.

Topical applications of 7,12-dimethylbenz(a)anthracene (DMBA) elicit a striking increase in melanogenically active melanocytes in both the dermis and the normally nonpigmented interfollicular epidermis of the adult trunk skin of the Mongolian gerbil. Although croton oil also produces marked hyperpigmentation of the dermis, it does not stimulate increased pigmentation of the epidermis. Light microscopy of the dorsal hairy skin of the gerbils revealed numerous dopa-positive melanocytes in the interfollicular epidermis at birth, but these declined sharply during the first month. Electron microscopy of the interfollicular epidermis of the adult gerbils demonstrated that Langerhans cells and indeterminate cells were the dominant nonkeratinocytes. Topical application of DMBA (once weekly with 1 ml of 0.1% DMBA in acetone) for 9 wk caused striking increases in melanogenically active epidermal melanocytes. The epidermis became heavily melanized. The melanocytes appeared damaged and may have become either dislodged from the basal layer or phagocytized by adjacent keratinocytes. There was a marked increase in dermal melanocytes and macrophages in DMBA-treated skin. Dermal blue nevus-like tumors elicited by DMBA contained cells laden with round to elliptical melanosomes. (20 refs.)

- 77-1918 **Retinyl Acetate Modulation of Cell Growth Kinetics and Carcinogen-Cellular Interaction in Mouse Epidermal Cell Cultures.** (Eng.) Yuspa, S. H. (In Vitro Pathogenesis Section, Experimental Pathology Branch, Div. Cancer Cause and Prevention, NCI, Bethesda, MD 20014) Elgjo, K.; Morse, M. A.; Wiebel, F. J. *Chem Biol Interact* 16(3): 251-264; 1977.

The exposure of newborn Balb/c mouse epidermal cell cultures to B-retinyl acetate (RA) was assessed. The binding of cellular protein was increased in the presence of RA, but the binding of 7,12-dimethylbenz(a)anthracene to epidermal cell DNA was decreased. DNA repair in response to chemical and physical agents was not affected. RA changed the course of cell differentiation. This resulted in a decreased rate of cell death that normally followed cellular maturation during the first 2 wk in culture. The stabilization of cell density by RA was most likely related to an extended life span of the cells. The level of aryl hydrocarbon hydroxylase (AHH) induced by benz(a)anthracene was strongly reduced to 20% of the controls after exposure to RA, but the activity of constitutive AHH was only slightly reduced. The studies suggest that retinoids may be interfering in a number of significant cellular functions involved in malignant transformation. (26 refs.)



**77-1919 Vulnerability of Specific Rat Chromosomes to In Vitro Chemically Induced Damage.** (Eng.)

Popescu, N. C. (Somatic Cell Genetics Section, Biology Branch, Carcinogenesis Program, Div. Cancer Cause and Prevention, NCI, Bethesda, MD 20014) DiPaolo, J. A. *Int J Cancer* 19(3): 419-433; 1977.

The acute effects of chemicals on chromosomes were studied in cultured rat embryo cells. 7,12-Dimethylbenz(a)anthracene (DMBA) or <sup>3</sup>H-DMBA administered 5 or 9 hr prior to the first mitosis or 24 hr prior to the second division resulted in chromatid-type aberration. The incidence of aberrations increased with length of exposure. The first two pairs of autosomes were involved preferentially; of these, the No. 2 pair, the largest telocentric chromosome, was affected more often. G-band analysis revealed four regions of chromosome No. 2 that were vulnerable to DMBA damage. All regions were associated with negative bands, and band 2q24 was the most vulnerable. In a few metaphases, chromosome No. 2 had dense <sup>3</sup>H-DMBA label approx covering the vulnerable breakage segment(s). The label associated with <sup>3</sup>H-DMBA was removable by digestion with DNase, suggesting that visible label represents cellular-bound carcinogen. After DMBA and bromodeoxyuridine (BUdR), there was an increase in the number of sister chromatid exchanges compared to controls treated with BUdR only. Experiments with <sup>3</sup>H-thymidine showed that the nonrandom chromatid lesions on chromosome No. 2 may result from endogenous radiation from incorporated <sup>3</sup>H. (41 refs.)

**77-1920 Post-replication Repair of 7-Bromomethyl Benz(a)anthracene Damaged DNA in Chinese**

**Hamster Cells (Meeting Abstract).** (Eng.) Roberts, J. J. (Inst. Cancer Res., Royal Cancer Hosp., Pollards Wood Res. Station, Nightingales Lane, Chalfont St. Giles, Bucks HP8 4SP, England) van den Burg, H. W.; Friedlos, F.; Kirkland, D. *Mutat Res* 46(2): 151; 1977. (no refs.)

**77-1921 7-Chloromethylbenz[a]anthracene.** (Eng.) Zacharias, D. E. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA 19111) *Acta Crystallogr [B] (Kbh)* B33(3): 902-905; 1977.

The molecular geometry of 7-chloromethylbenz[a]anthracene was determined. The structure was disclosed by the direct phasing method and refined using 1717 diffractometer data. The ring system was found to be essentially planar with a max atomic deviation of 0.05 Å. The C1 atom was located 1.47 Å above the plane and its attached C atom 0.16 Å below the plane. Data on crystal structure, bond lengths, and bond angles are presented. (9 refs.)

**77-1922 Synthesis and Properties of the Vicinal Trans Dihydrodiols of Anthracene, Phenanthrene, and Benzo(A)anthracene.** (Eng.) Lehr, R. E. (Natl. Inst. Arthritis, Metabolism, and Digestive Diseases, NIH, Bethesda, MD 20014) Schaefer-Ridder, M.; Jerina, D. M. *J Org Chem* 42(4): 736-744; 1977.

Chemical syntheses of the vicinal, trans, and non-K-region dihydrodiols of anthracene, phenanthrene, and benzo(a)anthracene (BA) are described. Discussion is made of the NMR spectra of the dihydrodiols and dihydrodiol esters and of the mutagenicity of the dihydrodiols and their metabolites toward *Salmonella typhimurium* strain TA100. None of the dihydrodiols synthesized were mutagenic per se, but 3,4-dihydroxy-3,4-dihydro-BA (9k) could be activated by metabolism to a strongly mutagenic species, and 8,9-dihydroxy-8,9-dihydro-BA and 10,11-dihydroxy-10,11-dihydro-BA could be activated to weakly mutagenic species. The greater mutagenicity of the activated 9k compound is in accord with the enhanced reactivity predicted by perturbational molecular orbital calculations for the benzylic positions of many intermediate diol epoxides in which the oxirane ring occupies a bay region. (31 refs.)

**77-1923 Protein Binding of Benz(a)anthracene and Benzo(a)pyrene.** (Eng.) Ma, J. K. (Sch. Pharmacy,

W. Virginia Univ., Morgantown, WV 26506) Fu, P. P.; Luzzi, L. A. *J Pharm Sci* 66(2): 209-213; 1977.

The noncovalent binding of benz(a)anthracene (BA) and benzo(a)pyrene (BP) to human serum albumin (HSA) was studied fluorimetrically. Each molecule of HSA was found to have one binding site for the hydrocarbons. At neutral pH, the binding constant for BA was  $1.56 \times 10^5 \text{ M}^{-1}$  and that for BP was  $1.46 \times 10^5 \text{ M}^{-1}$ . Studies of the fluorescence spectra indicated that both BA and BP bound to the same general area of HSA, but at different sites. BA bound 15.2 Å from the single tryptophan residue of HSA, and BP bound 19.6 Å from this residue. (19 refs.)

**77-1924 The Effect of Bile Acids on the Metabolism of Benzo(a)pyrene (BaP) and 2-Aminoanthracene**

**(2-AA) to Mutagenic Products (Meeting Abstract).** (Eng.) Kawalek, J. C. (Frederick Cancer Res. Center, Frederick MD 21701) Andrews, A. W. *Fed Proc* 36(3): 844; 1977. (no refs.)

**77-1925 Distribution of Covalently Bound Benzo(a)pyrene in Chromatin.** (Eng.) Jahn, C. L. (Corne

Univ. Graduate Sch. Medical Sciences, Rye, NY 10580) Litman, G. W. *Biochem Biophys Res Commun* 76(2): 534-540; 1977.

The distribution of covalently bound benzo(a)pyrene (BP) in nuclease-digestible and -undigestible regions of chromatin was studied. <sup>3</sup>H-BP was bound to the chromatin by incubating the carcinogen with calf thymus nuclei in the presence of NADPH and rat liver microsomes. Time-course digestion of the <sup>3</sup>H-BP-modified nuclei with Staphylococcal nuclease gave 50% acid-soluble products and resulted in a buildup of monomer-sized particles, followed by conversion of the monomer to smaller material. DNase I digestion gave 80% acid-soluble products and resulted in direct conversion to a size smaller than the monomer. Treatment with either enzyme decreased the specific activity of the undigested DNA. When DNA was extracted from the modified nuclei and digested with either enzyme, the undigested DNA had the same specific activity at all time points. Thus, BP appears to be preferentially located in digestible regions of DNA in chromatin. Kinetic studies of digestion by the two enzymes suggest that the BP binds primarily to the outermost "spacer" regions of the DNA. (24 refs.)

- 77-1926 **Eosinophilic Leukemia in a Syrian Hamster.** (Eng.) Port, C. D. (IIT Res. Inst., 10 W. 35th St., Chicago, IL 60616) Richter, W. R. *Vet Pathol* 14(3): 283-286; 1977.

A male Syrian golden hamster, part of a lifetime lung carcinogenesis bioassay using benzo(a)pyrene-hematite, was examined at death (at age 92 wk) for lung tumors. The tumor mass consisted of a scant loose stroma with many immature granulocytic cells. The lungs had extensive tumor infiltrates, the heart was partly surrounded by a large mass of tumor cells, the liver contained numerous periportal aggregations of these cells, and they had also infiltrated the intestine. The femoral marrow was extremely cellular and devoid of fat. To differentiate eosinophils from neutrophils, Luna's stain for eosinophil granules and a modification of the peroxidase reaction were used. However, results of both tests were equivocal. Several small blocks of affected liver were examined by transmission electron microscopy. The neoplastic cells in the liver were immature granulocytic cells, most with a round nucleus, although some had an obvious indented nucleus. There were cells with many large, round or oval, membrane-bound specific granules with dense, internal crystalloid structures of various shapes. These granules are a distinctive ultrastructural feature of eosinophils. It is suggested that this is the first report of eosinophilic leukemia in a laboratory animal. (12 refs.)

- 77-1927 **Covalent Binding of Benzo[A]pyrene to DNA and RNA at 1,3,6-Positions by Microsomal Activation.** (Eng.) Rogan, E. (Eppley Inst., Univ. Nebraska Medical Center, Omaha, NB 68105) Katomski, P.; Roth, R.; Cavalieri, E. *Fed Proc* 36(3): 305; 1977. (no refs.)

- 77-1928 **Metabolism of Benzo[a]pyrene (BP) in Cultured Human Bronchi (HB) and Pulmonary Macrophages (PAM) (Meeting Abstract).** (Eng.) Harris, C. (Dept. Pathology, Univ. Maryland, Baltimore, MD 21201) Autrup, H.; Stoner, G.; Trump, B.; Selkirk, J. *Fed Proc* 36(3): 305; 1977. (no refs.)

- 77-1929 **Experimental Evaluation of the Clearance of 3,4-Benzo(a)pyrene in Association with Talc from Hamster Lungs.** (Eng.) Pelfrene, A. F. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB 68105) *Am Ind Hyg Assoc J* 37(12): 706-710; 1977.

The effects of talc on the removal of benzo(a)pyrene (BP) from the lungs of Syrian golden hamsters were determined after intratracheal instillation of BP and talc as a saline suspension. After instillation of 3 mg BP alone, the percentages of applied dose recovered in the lungs were 59.3%, 56.1%, 29.5%, 5.7%, and 0% at time intervals of 0, 3, 6, 24, and 48 hr, respectively; each value is the av of data from six animals. With 3 mg of talc included in the instillate, the percentages of the same applied dose of BP recovered were 69.1%, 59.1%, 60.0%, 25.5%, 20.6%, 10.4%, and 0% after 0, 3, 6, 24, 48, 96, and 168 hr, respectively. With 9 mg of talc, the values were 79.8%, 65.5%, 62.6%, 38.1%, 24.1%, 5.3%, and 0% at the same time intervals. It is concluded that talc is an effective factor in retarding the clearance of BP from lung tissue. This may explain the known enhancement by talc of the tumor-inducing action of BP in the lung. (23 refs.)

- 77-1930 **New Metabolic Pathway of Benzo(a)pyrene 4,5-oxide, a Proximate Carcinogen of Benzo(a)pyrene (Meeting Abstract).** (Eng.) Kato, R. (Res. Lab., Fujisawa Pharmaceutical Co., Ltd., Osaka 532, Japan) Noguchi, H.; Iwasaki, K. *Jpn J Pharmacol* 26(Suppl): 86P; 1976. (no refs.)

- 77-1931 **Absolute Stereochemistry of the Highly Mutagenic 7,8-Diol 9,10-Epoxides Derived from the Potent Carcinogen *trans*-7,8-Dihydroxy-7,8-dihydrobenzo(a)pyrene (Letter to Editor).** (Eng.) Yagi, H. (Natl. Inst. Arthritis, Metabolism and Digestive Diseases, NIH, Bethesda, MD 20014) Akagi, H.; Thakker, D. R.; Mah, H. D.; Koreeda, M.; Jerina, D. M. *J Am Chem Soc* 99(7): 2358-2359; 1977.

The environmental carcinogen benzo[a]pyrene (BP) exerts most of its biologic activity by a covalent interaction between critical cellular entities and a particular class of reactive metabolites of the hydrocarbon, the diastereomeric 9,10-



epoxides of the benzo[a]pyrene-*trans*-7,8-dihydrodiol. These diastereomers were synthesized and assigned absolute configuration, and their individual biological activities were assessed. The assignment of absolute configuration was achieved by application of the exciton chirality circular dichroism (CD) method. The requirement of 7S, 8S-absolute stereochemistry was shown by the conversion of the diacetate of *trans*-7,8-dihydroxy-7,8,9,10-tetrahydrobenzo[a]pyrene in the presence of 10% Pd-C and a trace amount of concentrated HCl for 7 days. The diastereomeric 7,8-diol 9,10-epoxides were both found to be highly mutagenic. They alkylate the exocyclic N<sup>2</sup>-amino group of guanosine (G) in poly-G by *cis* and *trans* addition at C-10 and alkylate the phosphate backbone. When the skins of mice were treated with BP, it was discovered that both the *cis* and *trans* adducts of the diastereomeric (+)-7,8-diol 9,10-epoxides were important in the binding of BP to RNA *in vivo*. (22 refs.)

- 77-1932 **Metabolism of Benzo[a]pyrene. VI. Stereoselective Metabolism of Benzo[a]pyrene and Benzo[a]pyrene 7,8-Dihydrodiol to Diol Epoxides.** (Eng.) Thakker, D. R. (Natl. Inst. Arthritis, Metabolism and Digestive Diseases, NIH, Bethesda, MD 20014) Yagi, H.; Akagi, H.; Koreeda, M.; Lu, A. Y.; Levin, W.; Wood, A. W.; Conney, A. H.; Jerina, D. M. *Chem Biol Interact* 16(3): 281-300; 1977.

The resolution of ( $\pm$ )-benzo(a)pyrene 7,8-dihydrodiol (BP 7,8-dihydrodiol) into its (+)- and (-)-enantiomers is reported, and the specificity of hepatic cytochrome P-450 systems for the metabolism of both enantiomers to the highly mutagenic diol epoxide-1 and -2 is described. High stereoselectivity in the formation of diol epoxide-1 relative to diol epoxide-2 was noted with a purified cytochrome P-448-containing monooxygenase system and with liver microsomes from 3-MC-treated rats. The (+)-enantiomer produced a diol epoxide-2 to diol epoxide-1 ratio of 1:22, and the (-)-enantiomer produced a ratio of 6:1. The enantiomers were also metabolized to a phenolic derivative tentatively identified as 6,7,8-trihydroxy-7,8-dihydro BP. This compound represented approx 5% of the total metabolites with microsomes from 3-methylcholanthrene (3-MC)-treated Long-Evans-treated rats, but it accounted for approx 30% of the total metabolites formed by microsomes from phenobarbital-pretreated and control rats. With BP as substrate, liver microsomes produced the 4,5-, 7,8-, and 9,10-dihydrodiols with high optical purity; diol epoxides were also formed. Most of the optical activity in the BP 7,8-dihydrodiol was due to metabolism by the monooxygenase system rather than by epoxide hydrazase, since hydration of ( $\pm$ )-benzo[a]pyrene 7,8-oxide by liver microsomes produced dihydrodiol that was only 8% optically pure. The stereospecificity of both the monooxygenase system and, to a lesser extent, epoxide hydrazase is significant in the metabolic activation of BP to mutagens and carcinogens. (41 refs.)

- 77-1933 **Mechanism of Hydrolysis and Stereochemistry of the Hydrolysis Products and Their Acetonide of Two Stereoisomeric Benzo(a)pyrene 7,8-diol-9,10-Epoxides (Meeting Abstract).** (Eng.) Yang, S. K. (NIH, Bethesda, MD 20014) Roller, P. P.; Gelboin, H. V. *Fed Proc* 36(3): 844; 1977. (no refs.)

- 77-1934 **Benzo[a]pyrene Diol Epoxides: Mechanism of Enzymatic Formation and Optically Active Intermediates.** (Eng.) Yang, S. K. (Chemistry Branch, NCI, Bethesda, MD 20014) McCourt, D. W.; Leutz, J. C.; Gelboin, H. V. *Science* 196(4295): 119-1201; 1977.

Studies of the mechanism of benzo[a]pyrene (BP) metabolism to reactive diol epoxides are reviewed, and the detailed mechanism of the enzymatic hydration of racemic BP 7,8-epoxide is reported. The metabolic intermediates of the activation pathways, 7,8-epoxide and *trans*-7,8-diol, as well as the two stereoisomeric diol epoxides, were shown to be optically active. BP was converted to optically active 9,10-epoxides of (-)-*trans*-7,8-diol by three enzymatic steps: (1) stereospecific oxygenation at the 7,8 double bond of BP by the mixed-function oxidases to essentially a single enantiomer of 7,8-epoxide, (2) hydration of the 7,8-epoxide by epoxide hydrazase to an optically pure (-)-*trans*-7,8-diol, and (3) stereoselective oxygenation by the mixed-function oxidases at the 9,10 double bond of the (-)-*trans*-7,8-diol to optically active *r*-7,8-dihydroxy-*t*-9,10-oxy-7,8,9,10-tetrahydrobenzo[a]pyrene and optically active *r*-7,8-dihydroxy-*c*-9,10-oxy-7,8,9,10-tetrahydrobenzo[a]pyrene in a ratio of approximately 10 to 1. (41 refs.)

- 77-1935 **The Microsomal-Mediated Chemiluminescence of Benzo[a]pyrene Results from the Metabolism of 7,8-Dihydro-7,8-Dihydroxy Benzo[a]pyrene (Meeting Abstract).** (Eng.) Hamman, J. P. (Johns Hopkins Univ., Baltimore, MD 21218) Seliger, H. H. *Fed Proc* 36(3): 305; 1977. (no refs.)

- 77-1936 **Microsome-Induced Binding of Polycyclic Aromatic Hydrocarbons to Chromatin (Meeting Abstract).** (Eng.) Umans, R. S. (Boston Coll., Chestnut Hill, MA 02167) Mayo, K.; Boger, E. *Fed Proc* 36(3): 304; 1977. (no refs.)

- 77-1937 **A Bacteriophage System for the Screening and Study of Biologically Active Polycyclic Aromatic Hydrocarbons and Related Compounds (Meeting Ab-**

stract). (Eng.) Hsu, W. T. (The Franklin McLean Memorial Res. Inst., The Ben May Lab. for Cancer Res., Univ. Chicago, Chicago, IL 60637) Lin, E. J.; Harvey, R. G.; Weiss, S. B. *Fed Proc* 36(3): 304; 1977. (no refs.)

77-1938 Induction and Inhibition of the Formation of Benzo[a]Pyrene Metabolites That Covalently Bind to DNA by Inbred Mice (Meeting Abstract). (Eng.) Boobis, A. R. (NICHD, NIH, Bethesda, MD 20014) *Pharmacologist* 18(2): 210; 1976. (no refs.)

77-1939 Effect of  $\gamma$ -Hexachlorocyclohexane and Captane Ingestion on Benzo(a)pyrene Metabolism in Rat Liver (Meeting Abstract). (Eng.) Decloitre, F. (Institut de Recherches Scientifiques sur le Cancer, 94800-Villejuif, France) Mikol, Y. *Fed Proc* 36(3): 348; 1977. (no refs.)

77-1940 Inhibition of Skin Carcinogenesis by Diethylmaleate and Methionine Sulfoximine (Meeting Abstract). (Eng.) Chuang, A. H. (Univ. Vermont Coll. Medicine, Burlington, VT 05401) Mukhtar, H.; Bresnick, E. *Fed Proc* 36(3): 348; 1977. (no refs.)

77-1941 Differential Effects of Pre- Versus Post-Natal Exposure to 3-Methylcholanthrene (3-MC) on Hepatic and Lung Drug Metabolizing Activity and Tumorigenesis (Meeting Abstract). (Eng.) Soyka, L. F. (Dept. Pharmacology, Univ. Vermont Coll. Medicine, Burlington, VT 05401) Hunt, W. G.; Knight, S. E. *Fed Proc* 36(3): 305; 1977. (no refs.)

77-1942 Tumour Development after 3-Methylcholanthrene in Irradiated, Thymectomized Mice Reconstituted with Syngeneic Bone Marrow. (Eng.) Allegretti, N. (Dept. Physiology, Univ. Zagreb Faculty Medicine, Salata 3, 41001 Zagreb, p.o.b 978, Croatia, Yugoslavia) Marusic, M. *Eur J Cancer* 12(12): 1021-1024; 1977.

The induction of tumors in normal and in irradiated, thymectomized CBA mice by 3-methylcholanthrene (MC) was studied. The test animals were thymectomized and, 4 wk later, lethally irradiated with 900 R. Within 4 hr of irradiation the mice were reconstituted with  $1 \times 10^7$  normal syngeneic bone

marrow cells. MC was administered sc (1 mg in 0.1 ml of seed oil) 50 days later. Death and tumor production occurred, but tumor incidence did not differ significantly between T-cell deficient and normal mice. It was concluded that immunologically deficient mice have no greater susceptibility to tumor induction by MC than do normal mice and that the role of the thymus and the general immune surveillance system may be of little importance in counteracting malignancies. (18 refs.)

77-1943 Formation of 3-Methylcholanthrene Induced Cytochrome P-448 in Rats After Application of Cycloheximide (Meeting Abstract). (Eng.) Gerlach, R. (Dept. Pharmacology, Univ. Mainz, Obere Zahlbacher Strasse 67, D-6500 Mainz, W. Germany) Friederici, D. E.; Kahl, G. F. *Archiv fur Pharmakologie (Berlin)* 297(11, Suppl): R6; 1977. (no refs.)

77-1944 Evidence for the Catabolism of Polychlorinated Biphenyl-induced Cytochrome P-448 by Microsomal Heme Oxygenase, and the Inhibition of  $\delta$ -Aminolevulinic Acid Dehydratase by Polychlorinated Biphenyls. (Eng.) Maines, M. D. (Rockefeller Univ., New York, NY 10021) *J Exp Med* 144(6): 1509-1519; 1976.

The ability of cobalt to alter both the porphyrinogenic activity of polychlorinated biphenyls (PCB) and their cytochrome P448-inducing property was investigated, as was the possibility of degradation of PCB-induced cytochrome P448 by heme oxygenase. Pretreatment of rats with cobalt (250 micromoles/kg) 30 min before injection of 25 mg/kg sc PCB produced the following results on heme metabolism in liver: (1) augmentation of the porphyrinogenic effect of PCB, (2) augmentation of the PCB inhibition of  $\delta$ -aminolevulinic acid dehydratase (ALAD) activity; and (3) blockade of the induction of microsomal hemoprotein (cytochrome P448). When cobalt was administered 24 hr after PCB treatment, the magnitude of induction of  $\delta$ -aminolevulinic acid synthetase (ALAS) by PCB was lowered, and there was a large reduction in microsomal hemoprotein and heme contents. In the kidney, PCB blocked the induction of heme oxygenase and the depletion of cellular heme produced by cobalt. It is concluded that the heme moiety of microsomal cytochrome P448 is metabolized by the heme oxygenase system, that the synthesis of heme in the kidney and liver is regulated by different mechanisms, and that ionic cobalt controls the activity of ALAS by first inhibiting enzyme synthesis followed by indirect induction of the enzyme as a result of the catabolism of heme, the physiological repressor of ALAS, by the metal-induced heme oxygenase. (27 refs.)



77-1945 Metabolic Activation of [ $^{14}\text{C}$ ]Polychlorinated Biphenyls by Rat Liver Microsomes (Meeting Abstract). (Eng.) Shimada, T. (Osaka Prefectural Inst. Public Health, Higashinari-ku, Osaka 537, Japan) Iwagami, S. *Jpn J Pharmacol* 26(Suppl): 88P; 1976. (no refs.)

77-1946 Mutagenic Activity of Amino Acid Pyrolyzates in *Salmonella typhimurium* TA 98. (Eng.) Matsumoto, T. (Central Res. Inst., Japan Tobacco and Salt Public Corp., 6-2 Umegaoka, Midori-ku, Yokohama, Kanagawa 227, Japan) Yoshida, D.; Mizusaki, S.; Okamoto, H. *Mutat Res* 48(3/4): 279-286; 1977.

Pyrolyzates of 22 amino acids, 3 amino acid monohydrochloride, and 5 indole derivatives were tested for mutagenicity in the histidine-requiring mutants *Salmonella typhimurium* TA98 and TA100. Significant mutagenic activities were detected with the pyrolyzates of all amino acid monohydrochlorides and all amino acids except glycine, L-asparagine, L-aspartic acid, and L-histidine. The pyrolyzate of L-tryptophan had the highest mutagenic activity of the amino acids tested. The optimal pyrolysis temperatures for the formation of mutagenic products were 500 C for L-tryptophan and 600 C for the other amino acids. The pyrolyzates required a liver microsomal preparation to be detected as mutagens. The mutagen formed by pyrolysis of L-tryptophan was much more active toward TA98 than TA100, indicating that the pyrolyzate contained a frame-shift type mutagen. Experiments with the indole derivatives suggest the following relationships between molecular structure and pyrolyzate mutagenicity; (1) the mutagenic activity increases with carbon numbers of the substituent at the 3-position of the indole ring; and (2) the presence of an amino group at the  $\alpha$ -position to the carboxyl group of L-tryptophan plays an important role in the formation of mutagens. (18 refs.)

77-1947 Mutagenicity of Protein Pyrolysates. (Eng.) Nagao, M. (Biochemistry Div., Natl. Cancer Center Res. Inst., Tsukiji 5-1, Chuo-ku, Tokyo 104, Japan) Honda, M.; Seino, Y.; Yahagi, T.; Kawachi, T.; Sugimura, T. *Cancer Lett* 2(6): 335-340; 1977.

The mutagenicity of smoke condensates obtained by the pyrolysis of proteins and amino acids was studied using *Salmonella typhimurium* TA100 and TA98. Condensates from the pyrolysis of lysozyme and histone showed strong mutagenic activity on TA98, with metabolic activation (MA). The RNA showed low mutagenic activity with MA. Starch condensates showed a weak base-substituting mutagenic activity without MA. Vegetable oil condensates showed a slight mutagenic activity to TA100 with MA. When smoke condensates generated by the pyrolysis of various amino acids were subjected to mutation tests, tryptophan yielded the most potent activity. Kynurenine acid, a metabolic product of L-

tryptophan, yielded almost no mutagenic pyrolysis product. L-Tryptophan, D-tryptophan, 5-hydroxy-D,L-tryptophan, and kynurenine acid themselves were not mutagenic to either strain. Smoke condensates from chicken egg white lysozyme and calf thymus histone were both strongly mutagenic. Because of the high correlation between mutagenicity and carcinogenicity, it is suggested that the cooking of proteinaceous foods may be an important cause of human cancers. (12 refs.)

77-1948 In Vitro Inhibition of Aryl Hydrocarbon Hydroxylase by Heavy Metals. (Eng.) Tsang, S. (Inst. Chemical Biology, Univ. San Francisco, San Francisco, CA 94117) Furst, A. *Oncology* 33(5-6): 201-204; 1977.

The effect of carcinogenic and noncarcinogenic metal ions on aryl hydrocarbon hydroxylase (AHH) activity in mouse liver homogenates was determined. AHH was induced by an ip injection of 100 mg/kg 3-methylcholanthrene (3-MC). The mice were killed 24 hr later, their livers were removed, and homogenates were prepared. AHH activity was assayed by adding benzo(a)pyrene to the homogenates and determining the rate of appearance of 3-hydroxybenzo(a)pyrene (3-OH-BP). All five metals studied ( $\text{Ni}^{+2}$ ,  $\text{Cd}^{+2}$ ,  $\text{Zn}^{+2}$ ,  $\text{Mn}^{+2}$ , and  $\text{Fe}^{+2}$ ) showed some inhibition of AHH activity at concentrations as low as  $10^{-6}$  M. The inhibition increased as the metal concentration increased. When the concentrations of all five ions reached between  $10^{-2}$  and  $10^{-1}$  M, all AHH activity ceased. Of the five metals tested,  $\text{Cd}^{+2}$  showed the greatest inhibitory activity. Since both carcinogenic and noncarcinogenic metals showed inhibitory effects, it was concluded that the AHH system is not useful as an in vitro screen for carcinogenic metals. (18 refs.)

77-1949 The Effect of Cigarette Smoke on Aryl Hydrocarbon Hydroxylase Activity and Cytochrome P450 Content in Rat Liver and Lung Microsomes. (Eng.) Kushinsky, R. (Dept. Pathology, Univ. Melbourne, Austin Hosp., Heidelberg, Victoria 3084, Australia) Louis, C. J. *Oncology* 33(5-6): 197-200; 1976.

The inducibility of aryl hydrocarbon hydroxylase (AHH) activity in rat lungs exposed to graded doses of cigarette smoke was investigated. The levels of AHH activity in the human bronchial mucosa of smokers, nonsmokers, and patients with lung cancer were compared. Female Sprague-Dawley rats were used. Two groups of animals were injected ip with benzo(a)pyrene (BP) in maize oil at dose levels of 150 and 300 mg/kg, respectively. A third group received oil only and a fourth group received no injections. Corn oil produced no significant change in AHH activity in either liver or lung. After exposure to the smoke of four cigarettes, AHH activity increased sevenfold in the liver and threefold in the lung compared with that in untreated rats. Injection of 30 and 150 mg/kg BP elevated hepatic AHH activity 30 and 35 times

respectively, and lung AHH activity 9 and 6 times. Smoking in conjunction with BP injection resulted in lower levels of AHH activity in the lung than those from BP alone. These levels were not statistically different from those produced by smoking alone. Detectable levels of AHH activity were found in only 1/40 samples of human lung tissue, and that was from a 51-yr-old man who was a nonsmoker. Injection of corn oil increased cytochrome P450 levels in the liver microsomes. Smoking and/or 30 mg/kg BP also increased the amount of cytochrome P450, but only smoking alone and BP alone increased the amount significantly higher than that produced by oil alone. (23 refs.)

phases, the nitromethane phase, or a recombination of all fractions. (7 refs.)

- 77-1950 **Syncarcinogenic Action of Polycyclic Hydrocarbons in Automobile Exhaust Gas Condensates.** (Eng.) Schmahl, D.; Schmidt, K. G.; Habs, M. In: *Air Pollution and Cancer in Man. Proceedings of the Second Hanover International Carcinogenesis Meeting held in Hanover, 22-24 October, 1975.* Mohr, U.; Schmahl, D.; Tomatis, L.; Davis, W., eds. International Agency for Research on Cancer. (Lyon, France): IARC Scientific Publications No. 16 pp. 53-59; 1977.

Interactions between carcinogenic polycyclic aromatic hydrocarbons (PAH) and supposedly noncarcinogenic PAH in automobile exhaust fumes were investigated in NMRI mice. One group of animals was treated topically with a carcinogenic PAH mixture comprising 1, 1.7, or 3.0  $\mu\text{g}$  benzo(a)pyrene (BP) and equivalent proportions of dibenz(a,h)anthracene, benzo(a)anthracene, and benzo(b)fluoranthene. Two other groups were treated, respectively, with 7 noncarcinogenic PAH in amounts corresponding to 1, 3, and 27  $\mu\text{g}$  BP or with all 11 PAH at doses corresponding to those used in the first group. Tumors developed in mice receiving high doses of the noncarcinogenic PAH as well as in those treated with the carcinogenic compounds. In addition, the whole mixture was more effective than the carcinogenic compounds alone. (5 refs.)

- 77-1952 **Tumours in Mice After Subcutaneous Injection of Automobile Exhaust Condensate.** (Eng.) Pott, F.; Tomingas, R.; Misfeld, J. In: *Air Pollution and Cancer in Man. Proceedings of the Second Hanover International Carcinogenesis Meeting held in Hanover, 22-24 October, 1975.* Mohr, U.; Schmahl, D.; Tomatis, L.; Davis, W., eds. International Agency for Research on Cancer. (Lyon, France): IARC Scientific Publications No. 16 pp. 79-87; 1977.

Automobile exhaust condensate (AEC: 0.5 ml), mixed with benzo[a]pyrene (BP) or tricaprylin, was injected sc into NMRI mice in a series of experiments. The addition of AEC decreased the incidence of tumor that developed with 30, 90, and 270  $\mu\text{g}$  BP. Reduction of tumor incidence was proportional to the amount of AEC added. With an injection of 10  $\mu\text{g}$  BP, the latent period increased greatly when AEC was added, but the occurrence of tumors was the same. Components of AEC appear to inactivate BP, at least temporarily. In further experiments AEC and nine of its fractions were injected sc into mice. The fraction comprising only polycyclic aromatic hydrocarbons (PAH) induced the highest incidence of tumors. In contrast the PAH fraction was less active when administered in combination with other fractions. Application of the products of further fractionation of PAH showed that polycyclic compounds with seven or more rings can also induce tumors in this model. (11 refs.)

- 77-1953 **A New Type of Epoxide Hydratase Inducer (Meeting Abstract).** (Eng.) Schmassmann, H. U. (Inst. Pharmacology, Univ. Mainz, Obere Zahlbacher Str. 67, D-6500 Mainz, W. Germany) Sparrow, A.; Platt, K.; Oesch, F. *Archiv fur Pharmakologie (Berlin)* 297(11, Suppl): R7; 1977. (no refs.)

- 77-1951 **Experimental Results with Percutaneous Applications of Automobile Exhaust Condensates in Mice.** (Eng.) Brune, H. F. In: *Air Pollution and Cancer in Man. Proceedings of the Second Hanover International Carcinogenesis Meeting held in Hanover, 22-24 October, 1975.* Mohr, U.; Schmahl, D.; Tomatis, L.; Davis, W., eds. International Agency for Research on Cancer. (Lyon, France): IARC Scientific Publications No. 16 pp. 41-47; 1977.

The induction of squamous-cell carcinomas and papillomas in CFLP mice after painting their skin for 80 wk with different doses of automobile exhaust condensates and their fractions is described. The animals received 0.15, 0.60, or 1.80  $\mu\text{g}$ /treatment of the total condensate; 0.60 or 1.80  $\mu\text{g}$  of the methanol phase; and 0.30 or 0.90  $\mu\text{g}$  of the two cyclohexane

- 77-1954 **Cytochrome P-450-Mediated Drug Toxicity Studied in Isolated Hepatocytes (Meeting Abstract).** (Eng.) Moldeus, P. (Dept. Forensic Medicine, Karolinska Inst. 104 01 Stockholm, Sweden) Hogberg, J.; Orrenius, S. *Fed Proc* 36(3): 843; 1977. (no refs.)

- 77-1955 **Urological Aspects of Phenacetin (Acetophenetidin) Abuse.** (Ger.) Sage, S. (Urologischen Klinik der Medizinischen Akademie, Fetscherstrasse 74, DDR-8019 Dresden, E. Germany) *Z Urol Nephrol* 70(5): 351-355; 1977.



Studies of the side effects of phenacetin (acetophenetidin) abuse are discussed. Phenacetin is metabolized and excreted by the kidneys. It is metabolized to n-acetyl-p-aminophenol at a rate of about 99%, to p-phenetidin at a rate of 0.2%, then to p-hydroxylaminophenol by a cytochrome P-450-dependent mechanism, and, ultimately, to p-nitrosophenetol. N-hydroxylamines have cytotoxic and carcinogenic effects. An increased incidence of renal cell carcinoma and carcinoma of the renal pelvis and bladder was found among phenacetin-abuse patients. The tumors were often associated with chronic interstitial nephritis, another side effect of phenacetin. Tumor incidences were also compared with general autopsy findings from the prephenacetin era. A relationship was found between the occurrence of these carcinomas and the market volume of phenacetin-containing drugs, as well as between the incidences in men and women and the corresponding incidences of phenacetin abuse in both sexes. (17 refs.)

- 77-1956 **Mutagenicity Tests on Anthelmintics: Microsomal Activation of Vipryinium Embonate to a Mutagen.** (Eng.) Macphee, D. G. (Dept. Genetics, La Trobe Univ., Bundoora, Victoria, 3083 Australia) Podger, D. M. *Mutat Res* 48(3/4): 307-312; 1977.

The following eight anthelmintic preparations were tested for mutagenicity in the *Salmonella typhimurium* test system: Antepar (active ingredient, piperazine citrate), Combantrin (pyrantel embonate), Divermex (piperazine adipate), Hetrazan (diethylcarbamazine), Mintezol (thiabendazole), Pripsen (piperazine phosphate + senna), Vanquin (vipryinium embonate), and Yomesan (niclosamide). The test samples were placed in wells cut out of the agar of a plate previously seeded with a mutant strain of *S. typhimurium*. A mixture of rat liver microsomal enzymes and appropriate cofactors was added to one of two wells on a single plate to allow a possible requirement for metabolic activation to be recognized. None of the eight preparations was mutagenic by itself, but vipryinium embonate was activated by the rat liver microsomal preparation to a mutagen capable of causing both base-pair substitution and frameshift mutations. Further evaluation of the safety of this preparation is recommended, particularly because it is widely used by children and often by individuals who may not even be worm-infested (eg, the family of an affected individual). (8 refs.)

- 77-1957 **Inactivation of Carcinogens by Ozonation as Monitored by the Ames Mutagenesis Assay (Meeting Abstract).** (Eng.) Caulfield, M. J. (Univ. Notre Dame, Notre Dame, IN 46556) Burleson, G. R. *Fed Proc* 36(3): 1079; 1977. (no refs.)

- 77-1958 **Mutagens in Decomposition Products of Carbohydrates (Meeting Abstract).** (Eng.) Setliff, J.

A. (Pacific Biomedical Res. Center and Dept. Biochemistry and Biophysics, John A. Burns Sch. Medicine, Univ. Hawaii Honolulu, HI 96822) Mower, H. F. *Fed Proc* 36(3): 304; 1977. (no refs.)

- 77-1959 **Activation of Environmental Carcinogens by Human Liver Enzymes (Meeting Abstract)** (Eng.) Tang, T. (Dept. Microbiology, Virginia Commonwealth Univ., Richmond, VA 23298) Friedman, M. A. *Fed Proc* 36(3): 304; 1977. (no refs.)

- 77-1960 **Analysis of Dose-Effect Relationships of Carcinogens with a Median-Effect Principle (Meeting Abstract).** (Eng.) Chou, T. C. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021) *Fed Proc* 36(3): 304; 1977. (no refs.)

- 77-1961 **Alkaline Labile DNA in Rat Tissues Following Administration of Three Carcinogens and/or Mutagens (Meeting Abstract).** (Eng.) Lilja, H. S. (Dept. Pathology, Dartmouth Medical Sch., Hanover, NH 03755) Culphey, T. J.; Hyde, E.; Longnecker, D. S.; Yager, J. D. *Fed Proc* 36(3): 305; 1977. (no refs.)

- 77-1962 **Chemical Carcinogen and Lipoperoxidative Effects on the Ribosome Membrane Complex of Rough Microsomal Membranes (Meeting Abstract).** (Eng.) Shires, T. K. (Toxicology Center, Dept. Pathology, Univ. Iowa, Iowa City, IA 52242) *Fed Proc* 36(3): 1078; 1977. (no refs.)

\* (Review): 77-1801, 77-1802, 77-1803, 77-1804, 77-1806, 77-1807, 77-1808, 77-1809, 77-1810, 77-1811, 77-1814, 77-1815, 77-1816, 77-1817, 77-1818, 77-1819, 77-1834, 77-1835, 77-1836, 77-1837, 77-1846.

\* (Phys): 77-1974, 77-1977, 77-1988, 77-1995, 77-1997, 77-2002, 77-2005.

\* (Viral): 77-2039, 77-2040, 77-2068.

\* (Immun): 77-2075, 77-2088, 77-2095, 77-2096, 77-2101, 77-2107, 77-2114, 77-2130, 77-2144, 77-2162, 77-2163, 77-2217, 77-2218, 77-2220, 77-2232.

\* (Path): 77-2246, 77-2253, 77-2259, 77-2266, 77-2267.

\* (Epid): 77-2292, 77-2293, 77-2302, 77-2303, 77-2304, 77-2306, 77-2308.

## PHYSICAL CARCINOGENESIS

- 77-1963 **The Effect of Ultraviolet Light on Arrested Human Diploid Cell Populations.** (Eng.) Kantor, G. J. (Dept. Biological Sciences, Wright State Univ., Dayton, OH 45431) Warner, C.; Hull, D. R. *Photochem Photobiol* 25(5): 483-489; 1977.

The results of initial experiments to determine the effects of UV light (254 nanometers) on human diploid fibroblast (HDF) arrested in a nondividing stage are reported. A fraction of the arrested cells did not remain attached to a plastic culture surface following exposure to UV light. Dye-exclusion tests and unsuccessful attempts to culture the detached cells indicated that they were not viable. These cells were fluence-dependent, with the percentage of survival decreasing as the fluence increased. The age of the HDF populations had no significant effect on survival following irradiation, but the length of the preirradiation arrested period did. The longer the arrested period, the more resistant the cells became. Xeroderma pigmentosum cells were more sensitive to UV light than normal HDF, and survival was not affected by changing the length of the preirradiation arrested period. The results indicate that DNA repair plays a role in maintaining irradiated cells in the arrested state and that the lethal event caused by UV irradiation affects transcription, leading to an inhibition of required protein synthesis. (30 refs.)

- 77-1964 **Antioxidant-mediated Reversal of Ultraviolet Light Cytotoxicity.** (Eng.) Chan, J. T. (Dept. Dermatology, Baylor Coll. Medicine, Texas Medical Center, Houston, TX 77025) Black, H. S. *J Invest Dermatol* 68(6): 366-368; 1977.

Ascorbic acid [0.01 micromole ( $\mu\text{mol}$ )/ml], glutathione (0.02  $\mu\text{mol}$ /ml), DL- $\alpha$ -tocopherol (0.002  $\mu\text{mol}$ /ml), and butylated hydroxytoluene (0.02  $\mu\text{mol}$ /ml) were tested for their ability to protect Chinese hamster embryo cells against UV light meters, 0.5 millijoule/cm<sup>2</sup>). All four antioxidants reversed UV-induced cytotoxicity when added to the culture medium immediately after irradiation; the colony-forming ability of irradiated cells incubated with the antioxidants for 8 days increased by > 100%. At the concentrations tested, no protective effect was detected when the antioxidants were added prior to UV light irradiation. (16 refs.)

- 77-1965 **UV-Light Induced Sister Chromatid Exchanges in Xeroderma Pigmentosum Lymphocytes.** (Eng.) Schonwald, A. D. (Institut für Humangenetik, Uni-

versität Hamburg, D-2000 Hamburg, W. Germany) Passarge, E. *Hum Genet* 36(2): 213-218; 1977.

Cultured lymphocytes from nine patients (4 men and 5 women aged 12-38 yr) with clinically different types of xeroderma pigmentosum were exposed to UV light at 24 hr. The rate of sister chromatid exchanges was normal in xeroderma lymphocytes not exposed to UV light. However, it increased in six patients after the cultures were irradiated two cell cycles before harvest. Three types of response could be differentiated: an increase of 128%-148% in three, 34%-51% in three, and no increase in three patients with deSanctis-Cacchione Syndrome. Both the dose and timing of UV irradiation seemed to be significant. Cultures irradiated at 0, 48, or 72 hr differed less clearly from the controls. The concomitant control cultures did not respond with an increased rate of exchanges. No evidence for an increased rate of chromatid breaks was observed in any of the cultures. An obligate heterozygote, the daughter of one of the patients, had a sister chromatid exchange rate of 9.0 prior to UV exposure and an unchanged rate of 11.7 following UV exposure. The expression of UV light-induced chromosomal instability may be due to impaired DNA repair. (16 refs.)

- 77-1966 **Evidence for the Generation of Suppressor Cells by Ultraviolet Radiation.** (Eng.) Daynes, R. A. (Dept. Pathology, Univ. Utah Medical Center, Salt Lake City, UT 84132) Spellman, C. W. *Cell Immunol* 31: 182-187; 1977.

Most murine skin tumors induced by UV light are unique in that they are of sufficient antigenicity to be consistently rejected when transplanted into normal syngeneic animals. However, the exposure of normal syngeneic mice to subcarcinogenic levels of UV prior to tumor transfer results in the progressive growth of transplanted UV tumors. Normal C3Hf/He female mice were also rendered tumor-susceptible by the adoptive transfer of lymphoid cells from either tumor-bearing or short-term UV exposed donors. Further, the adoptive transfer of tumor susceptibility could be abolished by the pretreatment of cell suspensions from UV-exposed donors with anti-theta and complement. These results suggest that UV irradiation may generate the development of T lymphocytes with suppressor activity. (9 refs.)

- 77-1967 **Introduction of Sister Chromatid Exchanges in Xeroderma Pigmentosum Cells Following UV Exposure (Meeting Abstract).** (Eng.) de Weerd-Kastelein, E. A. (Dept. Cell Biology and Genetics, Erasmus Univ., Rotter-



dam, Netherlands) Keijzer, W.; Rainaldi, P. *Mutat Res* 46(2): 163; 1977. (no refs.)

77-1968 A Study of Postreplication Repair Mechanisms in Xeroderma Pigmentosum Fibroblast Cells (Meeting Abstract). (Eng.) Minka, D. F. (West Virginia Univ., Morgantown, WV 26506) *Diss Abstr Int [B]* 37(12/ Part 1): 5968-5969; 1977. (no refs.)

77-1969 5-Bromodeoxyuridine Protection from Ultraviolet Damage in UV-Endonuclease Deficient Cells (Meeting Abstract). (Eng.) Carlson, K. M. (Univ. Miami, Coral Gables, FL 33124) *Diss Abstr Int [B]* 37(12/ Part 1): 5977; 1977. (no refs.)

77-1970 Simian Virus 40 (SV40) DNA Synthesis in UV-Irradiated Monkey CV-1 Cells (Meeting Abstract). (Eng.) Williams, J. I. (Lab. Radiobiology, Univ. California at San Francisco, CA 94122) Cleaver, J. E. *Radiat Res* 70(3): 667-668; 1977. (no refs.)

77-1971 Ultraviolet Induction of Leukemia Virus from Mouse Cells (Meeting Abstract). (Eng.) Hellman, K. B. (Bureau Radiological Health, Food and Drug Admin., Dept. Health, Education and Welfare, Rockville, MD 20857) Brewer, P. P.; Hellman, A. *Radiat Res* 70(3): 667; 1977. (no refs.)

77-1972 Reduced Host Cell Reactivation of UV-irradiated Adenovirus in Fanconi's Anaemia Fibroblasts (Meeting Abstract). (Eng.) Rainbow, A. J. (Dept. Radiology, McMaster Univ., Hamilton, Ontario L8S 4J9, Canada) Howes, M. *Radiat Res* 70(3): 686; 1977. (no refs.)

77-1973 Studies on the Kinetics of Thymine Dimer Excision in Human Cells In Vivo (Meeting Abstract). (Eng.) Ehmann, U. K. (Dept. Pathology, Stanford Univ., Stanford, CA 94305) Friedberg, E. C. *Radiat Res* 70(3): 686; 1977. (no refs.)

77-1974 Chromosome Damage in Chinese Hamster Cells Sensitized to Near-ultraviolet Light by Psoralen and Angelicin. (Eng.) Ashwood-Smith, M. J. (Dept. Biology,

Univ. Victoria, Victoria, British Columbia, Canada V8W 2Y2) Grant, E. L.; Heddle, J. A.; Friedman, G. B. *Mutat Res* 43(3): 377-385; 1977.

The clastogenic effect of the two furocoumarins in the presence of near-UV differs greatly, as do their modes of interaction with DNA. Psoralen, which requires only one-fifth as much light energy to produce the same lethal effect as angelicin at equimolar concentrations, is able to cross-link DNA as well as to form monoadducts; but angelicin is only capable of monoadduction. Comparable numbers of micronuclei at comparable survivals are produced despite the differences in cross-linking ability and resultant differences in lethality. Chromosomal aberrations account for much or all of the lethality observed. Metaphase analysis at comparable aberration frequencies revealed that angelicin and psoralen both induce chromatid deletions and a wide spectrum of chromatid exchanges. These data show that both cross-links and monoadducts to the DNA can result in chromosomal aberrations. The relative contributions of cross-links and monoadducts to chromosomal aberrations still remain to be determined. Extensive chromosomal damage can be induced in mammalian cells by the combination of psoralen and near-UV, a treatment that is currently widely used in the therapy of psoriasis. (15 refs.)

77-1975 Endonuclease-Sensitive Sites Induced by Fluorescent Light (Meeting Abstract). (Eng.) Ritter, M. A. (Harvard Sch. Public Health, Boston, MA 02115) Williams, J. R. *Radiat Res* 70(3): 668; 1977. (no refs.)

77-1976 BUdR and Fluorescent Light-Induced Mutagenesis in Synchronous Chinese Hamster Cells In Vitro (Meeting Abstract). (Eng.) Burki, H. J. (Biology and Medicine Div., Lawrence Berkeley Lab., Univ. California, Berkeley, CA 94720) *Radiat Res* 70(3): 650; 1977. (no refs.)

77-1977 Screening for the Mutagenicity of Nitro-Group Containing Hypoxic Cell Radiosensitizers Using the Ames Assay (Meeting Abstract). (Eng.) Rauth, A. M. (Physics Div., Ontario Cancer Inst., 500 Sherbourne Street, Toronto, Ontario, Canada M4X 1K9) Chin, J.; Sheinin, D. *Radiat Res* 70(3): 701; 1977. (no refs.)

77-1978 Oncogenic Transformation In Vitro by X-Ray and the Hypoxic Sensitizer Ro-07-0582 (Meeting Abstract). (Eng.) Miller, R. C. (Columbia Univ., New York, NY 10032) Hall, E. J. *Radiat Res* 70(3): 649; 1977. (no refs.)

- 77-1979 **A Radiation-induced Pharyngeal Cancer.** (Jpn.) Sakai, K. (Dept. Radiology, Niigata Univ. Sch. Medicine, Niigata, Japan) Kurokawa, H.; Hinata, H.; Kitabatake, T.; Fukase, M.; Koizumi, F. *Jpn J Cancer Clin* 23(3): 220-224; 1977.

The case history of a 63-yr-old woman with pharyngeal cancer is presented. The cancer developed approx 37 yr after treatment with 7,600 rads of radiation for cervical tuberculous lymphadenitis. Biopsy showed squamous cell carcinoma. (22 refs.)

- 77-1980 **Genetic Damage from Diagnostic Radiation.** (Eng.) Bross, I. D. (Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263) Natarajan, N. *JAMA* 237(22): 2399-2401; 1977.

The working hypothesis that in utero exposure to diagnostic radiation produces both indicator diseases and leukemia was investigated. The hypothesis was tested by a new method for analyzing the Tri-State Survey data. This method was based on a mathematical model that described the population at risk and embodied the working hypothesis under test in mathematical language. There was an estimated 50-fold increase in the risk of leukemia and a 5-fold increase in certain other diseases for approx 1% of the exposed persons who are affected by radiation. These data on exposure to ordinary diagnostic radiation show that even when the radiation dose is fractionated, genetic damage occurs. (6 refs.)

- 77-1981 **Radiation Exposure and Thyroid Cancer.** (Eng.) Greenspan, F. S. (Room U 125, Univ. California, San Francisco, CA 94143) *JAMA* 237(19): 2089-2091; 1977.

The clinical characteristics of 94 patients with a history of either radiation exposure without known thyroid disease or radiation exposure and thyroid enlargement were reviewed. Forty-six patients had thyroid cancer, 48 did not. Cancer was found 5-40 yr after exposure. Of the 46 cancer patients, 7 also had adenomas, 3 had Hashimoto's thyroiditis, and 2 had both. Of the 48 patients without thyroid cancer, no thyroid disease or any other disease was found in 18; the rest had a variety of thyroid disorders, the most common lesions being multiple adenomas (12 patients) and Hashimoto's thyroiditis (8). Published data indicate that the incidence of thyroid cancer increases with increasing doses of thyroid radiation from 6.5 to 1,500 rads, but higher doses tend to destroy the gland and are associated with hypothyroidism rather than cancer. If there is thyroid enlargement without nodularity, hypothyroidism, or a strongly positive serum antithyroid antibody test, the patient should receive thyroxine therapy for life. Indications for surgery include a discrete, firm thyroid nodule that is cold on scan and the persistence of discrete, firm

nodules after a trial of thyroxine therapy for multinodular goiters. (17 refs.)

- 77-1982 **Nuclear Envelope-DNA Interaction in X-Irradiated CHO Cells (Meeting Abstract).** (Eng.) Blackburn, G. R. (Dept. Radiobiology and Radiation Biology, Colorado State Univ., Fort Collins, CO 80523) Highfield, D. P.; Dewey, W. C. *Radiat Res* 70(3): 640; 1977. (no refs.)

- 77-1983 **Differential Spermatogonial Stem Cell Survival and Mutation Frequency (Meeting Abstract).** (Eng.) Oakberg, E. F. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Palatinus, D. T. *Radiat Res* 70(3): 628-629; 1977. (no refs.)

- 77-1984 **Histological Changes in X-Radiated Salivary Glands of the Rhesus Monkey (Meeting Abstract).** (Eng.) Bowers, D. E. (Saint Louis Univ., Saint Louis, MO 63103) *Diss Abstr Int [B]* 37(12/Part 1): 5913; 1977. (no refs.)

- 77-1985 **The Magnitude and Nature of Late Effects of Radiation Injury in Abdominally Exposed or Shielded Mice (Meeting Abstract).** (Eng.) Spalding, J. F. (Mammalian Biology Group, Los Alamos Scientific Lab., Univ. California, Los Alamos, NM 87545) Archuleta, R. F.; Prine, J. R.; Wood, R. H. *Radiat Res* 70(3): 669; 1977. (no refs.)

- 77-1986 **The Thymus as a Modifier of Late Somatic Effects of X-radiation: Preliminary Observations on X-irradiated and Aging Control Germfree Athymic Nude Mice (Meeting Abstract).** (Eng.) Holland, J. M. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) *Radiat Res* 70(3): 669; 1977. (no refs.)

- 77-1987 **Dose-Effect Curve of X-Ray Induced Dominant Lethals in Post-Meiotic Germ Cells of Male Mice (Meeting Abstract).** (Eng.) Dunn, G. R. (Dept. Radiation Therapy, Harvard Medical Sch., Boston, MA 02115) *Radiat Res* 70(3): 629; 1977. (no refs.)



- 77-1988 **Radiation Dose-Response of Hepatic Aryl Hydrocarbon Hydroxylase in Mice (Meeting Abstract).** (Eng.) Prasad, R. (Baylor Coll. Medicine, Houston, TX 77030) Prasad, N.; Bushong, S. C.; Harrell, J. E. *Radiat Res* 70(3): 605-606; 1977. (no refs.)
- 77-1989 **Radiation Carcinogenesis in the Rat Colon (Meeting Abstract).** (Eng.) Kirchner, F. R. (Radiation Res. Lab., Dept. Radiology, Univ. Iowa, Iowa City, IA 52242) Denman, D. L.; Osborne, J. W. *Radiat Res* 70(3): 606-607; 1977. (no refs.)
- 77-1990 **Radiation-induced Oncogenic Transformation of Cultured Mouse Embryo Cells (Meeting Abstract).** (Eng.) Han, A. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439) Elkind, M. M. *Radiat Res* 70(3): 650; 1977. (no refs.)
- 77-1991 **Influence of Repair Processes on X-ray Induced Malignant Transformation In Vitro (Meeting Abstract).** (Eng.) Little, J. B. (Harvard Sch. Public Health, Boston, MA 02115) *Radiat Res* 70(3): 705-706; 1977. (no refs.)
- 77-1992 **Effects of Hyperthermia and X-Irradiation on Sister Chromatid Exchange Frequency in Chinese Hamster Ovary (CHO) Cells (Meeting Abstract).** (Eng.) Livingston, G. K. (Univ. Utah, Salt Lake City, UT 84132) Dethlefsen, L. A. *Radiat Res* 70(3): 611-612; 1977. (no refs.)
- 77-1993 **Hyperthermia and Radiation Induced Genetic Aberrations in *Drosophila* (Meeting Abstract).** (Eng.) Mittler, S. (Northern Illinois Univ., DeKalb, IL 60115) *Radiat Res* 70(3): 729-730; 1977. (no refs.)
- 77-1994 **Dose-effect Relationships for Tumour Induction by Radiation and the Effectiveness of Small Doses (Meeting Abstract).** (Eng.) Barendsen, G. W. (Radiobiological Inst. TNO, Rijswijk, Netherlands) *Int J Radiat Biol* 31(4): 380; 1977. (no refs.)
- 77-1995 **Interactions of Butylated Hydroxytoluene and Diethylnitrosamine in Modifying Radiation-Induced Lifeshortening and Tumorigenesis (Meeting Abstract).** (Eng.) Clapp, N. K. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Satterfield, L. C.; Klima, W. C. *Radiat Res* 70(3): 608; 1977. (no refs.)
- 77-1996 **The Combined Carcinogenic Action of Ionizing Radiation and DMBA on Rat Skin (Meeting Abstract).** (Eng.) Burns, F. J. (Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY 10016) Strickland, P.; Albert, R. E. *Radiat Res* 70(3): 607; 1977. (no refs.)
- 77-1997 **An Investigation into the Mechanism by Which Radiation or Cyclophosphamide Increases Blood-Borne Metastases in Mice (Meeting Abstract).** (Eng.) Brown, J. M. (Dept. Radiology, Stanford Univ., Stanford, CA 94305) *Radiat Res* 70(3): 689-690; 1977. (no refs.)
- 77-1998 **Limited Capacity of Chromosomal Proteins to Protect Eukaryotic DNA from Damage by Ionizing Radiation (Meeting Abstract).** (Eng.) Ansevin, A. T. (Univ. Texas System Cancer Center, M. D. Anderson Hosp., Houston, TX 77030) *Radiat Res* 70(3): 616; 1977. (no refs.)
- 77-1999 **Mutation Induction in Vegetative and Developing Cells of the Slime Mold, *Dictyostelium discoideum* (Meeting Abstract).** (Eng.) Deering, R. A. (Biochemistry and Biophysics, Pennsylvania State Univ., University Park, PA 16802) Sheely, M. *Radiat Res* 70(3): 649; 1977. (no refs.)
- 77-2000 **Early Malignant Neoplasms in Dogs Irradiated During the Perinatal Period (Meeting Abstract).** (Eng.) Phemister, R. D. (Collaborative Radiological Health Lab., Colorado State Univ., Fort Collins, CO 80523) Thomassen, R. W. *Radiat Res* 70(3): 669-670; 1977. (no refs.)
- 77-2001 **Enhancement of Viral Transformation of Cells In Vitro by Heavy Ion Radiation (Meeting Abstract).** (Eng.) Yang, T. C. (Donner Lab., Univ. California, Berkeley, CA 94720) Risius, J.; Madfes, I.; Tobias, C. A.; Lyman, J.; Howard, J. *Radiat Res* 70(3): 650-651; 1977. (no refs.)

- 77-2002 **Mammary Carcinogenesis in the Rat: Hormones and LET Effects (Meeting Abstract).** (Eng.) Clifton, K. H. (Dept. Human Oncology, Radiology and Statistics, Wisconsin Clinical Cancer Center, Univ. Wisconsin Medical Sch., Madison, WI 53706) Crowley, J. J. *Radiat Res* 70(3): 607; 1977. (no refs.)
- 77-2003 **Reduced Life-Shortening Effect of Fractionated Neutron or Gamma Doses Received Late in Life (Meeting Abstract).** (Eng.) Ainsworth, E. J. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439) Fry, R. J.; Williamson, F. S.; Allen, K. H.; Hulesch, J. S.; Jordan, D. L.; Kickels, W. T. *Radiat Res* 70(3): 608-609; 1977. (no refs.)
- 77-2004 **Age-Dependency of Radiation-Induced Late Effects (Meeting Abstract).** (Eng.) Fry, R. J. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439) Ainsworth, E. J.; Williamson, F. S.; Allen, K. H.; Ludeman, V. A.; Staffeldt, E. F.; Lombard, L. S. *Radiat Res* 70(3): 609; 1977. (no refs.)
- 77-2005 **Mammary Neoplastic Response to DES Administration Before or After Irradiation with 0.43 MeV Neutrons (Meeting Abstract).** (Eng.) Stone, J. P. (Medical Dept., Brookhaven Natl. Lab., Upton, NY 11973) Holtzman, S.; Shellabarger, C. J. *Radiat Res* 70(3): 607-608; 1977. (no refs.)
- 77-2006 **Neutron Carcinogenesis: Dose and Dose Rate Effects in BALB/c Mice (Meeting Abstract).** (Eng.) Ullrich, R. L. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Jernigan, M. C. *Radiat Res* 70(3): 608; 1977. (no refs.)
- 77-2007 **Incidence of Mammary Tumours in Rats of Different Strains After Fast Neutron Irradiation (Meeting Abstract).** (Eng.) Broerse, J. J. (Radiobiological Inst. TNO, Rijswijk, The Netherlands) Knaan, S.; van Bekkum, D. W.; Nooteboom, A. L.; Hollander, C. F. *Int J Radiat Biol* 31(4): 378-379; 1977. (no refs.)
- 77-2008 **Oncogenic Transformation In Vitro by  $\alpha$  Particles (Meeting Abstract).** (Eng.) Lloyd, E. L. (Argonne Natl. Lab., Argonne, IL 60439) Gemmell, A.; Henning, C. B.; Zabransky, B. J. *Radiat Res* 70(3): 648-649; 1977. (no refs.)
- 77-2009 **Early Effects of Single and Fractionated Doses of High Energy  $^{40}\text{Ar}$  Ions on the Skin of Rats (Meeting Abstract).** (Eng.) Strickland, P. T. (Inst. Environmental Medicine, New York Univ. Medical Center, New York, NY 10016) Burns, F. J.; Albert, R. E. *Radiat Res* 70(3): 673; 1977. (no refs.)
- 77-2010 **Effects of  $^{239}\text{Pu}$  on Hematopoiesis in Prenatal Rats (Meeting Abstract).** (Eng.) Joshima, H. (Biology Dept., Battelle, Pacific Northwest Lab., Richland, WA 99352) Hackett, P. L.; Sikov, M. R. *Radiat Res* 70(3): 622; 1977. (no refs.)
- 77-2011 **Radiation-Induced Chromosome Aberrations in Syrian Hamster Lung Cells by Inhalation of  $^{238}\text{PuO}_2$ - $\text{ZrO}_2$  (Meeting Abstract).** (Eng.) Stroud, A. N. (Mammalian Biology Group, Los Alamos Scientific Lab., Univ. California, Los Alamos, NM 87545) *Radiat Res* 70(3): 628; 1977. (no refs.)
- 77-2012 **Genetic Effects from Exposure of Male Mice to Tritium for Six Generations (Meeting Abstract).** (Eng.) Mewissen, D. J. (Univ. Chicago, Chicago, IL 60637) Ugarte, A. S.; Rust, J. H. *Radiat Res* 70(3): 629; 1977. (no refs.)
- 77-2013 **Numerical Evaluation of Asbestos Fibers in Wine Samples.** (Fre.) Bignon, J. (Service Hos-



pitalo-Universitaire de Pneumologie, Centre Hospitalier Intercommunal, 40, av. de Verdun, F 94010 Creteil, France) Bientz, M.; Bonnaud, G.; Sebastien, P. *Nouv Presse Med* 6(13): 1148-1149; 1977.

The risk of gastrointestinal tract cancer from ingestion of asbestos remains in dispute. However, workers exposed to asbestos have a higher incidence of gastrointestinal cancer, and recent studies in rats show a significant increase in tumor of various organs after ingestion of a diet with asbestos fibers. Since wine is usually clarified by methods incurring the risk of asbestos contamination, 22 random samples of wine were examined for asbestos fibers. Suspended particles in the samples were concentrated by ultrafiltration on a millipore filter and then observed under the electron microscope. Each fibrous particle was subjected to a morphological analysis and

a crystallographic study by electronic microdiffraction in order to establish it as an asbestos fiber. Of the 22 samples, 9 had asbestos fibers ranging in concentration from 2 to  $64 \times 10^6$  fibers/liter. Three samples of beer, also studied, did not contain asbestos. The relationship of method of filtration to asbestos particle contamination is under study. (7 refs.)

\* (Review): 77-1811, 77-1812, 77-1819.

\* (Chem): 77-1854.

\* (Viral): 77-2059.

\* (Immun): 77-2119, 77-2122, 77-2154, 77-2218.

\* (Path): 77-2262.

\* (Epid): 77-2298, 77-2299, 77-2300, 77-2301, 77-2303.

## VIRAL CARCINOGENESIS

77-2014 **Endogenous Leukosis Viruses in the Avian Family Phasianidae.** (Eng.) Chen, Y. C. (Dept. Microbiology, Univ. Southern California, Sch. Medicine, 2025 Zonal Ave., Los Angeles, CA 90033) Vogt, P. K. *Virology* 76(2): 740-750; 1977.

New endogenous viruses from several species of the family Phasianidae, which includes pheasants, quail, partridge, and the domestic chicken, are described. Some of the viruses are produced spontaneously, others are recombinants with Rous sarcoma virus (RSV). The defective Bryan high-titer strain of RSV without helper virus, RSV(-), was fused with  $\beta$ -propiolactone-inactivated Sendai virus into embryo fibroblast cultures of the avian species. The viruses used were Rous-associated virus type 1 (RAV-1) of subgroup A, RAV-6 of subgroup B, RAV-7 of subgroup C, RAV-0 and RAV-60 of subgroup E, ringneck pheasant virus (RPV), RAV-61 of subgroup F, and Carr-Zilber-associated virus (CZAV) of subgroup D. Chick helper factor (Chf)-positive and -negative chick embryo fibroblasts were used as control. Within 7 days after infection, foci of transformation were visible in the fused cultures. Pseudotypes of infectious RSV were recovered from RSV(-)-transformed cells of all species studied except duck, goose, Japanese quail, and pigeon. Chinese quail, chukar, and pheasants can produce viral glycoproteins that undergo phenotypic mixing with RSV(-) particles. Only one new pseudotype, from Chinese quail, induced foci on fibroblasts from the European field vole (*Microtus agrestis*) and on 3T3 clone A31 from Balb/c mice with high efficiency. Polybrene, a polycation, was used throughout, as it enhanced foci formation significantly. Helper virus activity in the Ghghi and silver pheasant has G envelope specificity; the activity in the green pheasant has F envelope specificity. No information is available on the pathogenic potential of any of these new viruses. (24 refs.)

77-2015 **The Study of the Rous Virus Synthesis in Mammalian Embryonic Tissues Growing In Vitro.** (Eng.) Shevlyaghin, V. J. (Gamaleya Inst. Epidemiology and Microbiology, Acad. Medical Sciences USSR, Moscow D-98 USSR) Chizhevskaya, V. *Folia Biol (Praha)* 23(2): 140-145; 1977.

Synthesis of the genome of Rous sarcoma virus (Schmidt-Ruppin strain) in untransformed mammalian cells was investigated. As determined by the appearance of tumors in chickens inoculated with living cells, the virus genome was present in mouse and hamster embryo cells growing in vitro shortly after infection and long before cell transformation. The virus genome was not detected in embryo cells treated with actinomycin D or when the incubation temperature was lowered from 37 to 20 C. This suggested that the Rous virus genome

was synthesized de novo in the cells. Furthermore, the experiments showed that the life cycle of Rous virus in mammalian and avian cells had at least one phase in common, the phase sensitive to the action of actinomycin D. (10 refs.)

77-2016 **Tumor Growth and Antibodies after RSV-Challenge in Normal Chickens and in Chickens Congenitally Infected with Avian Leukosis Virus.** (Eng.) Meyers, P. (Dept. Microbiology, Mayo Clinic, Rochester, MN 55901) Qualtiere, L. F. *J Immunol* 118(5): 1541-1548; 1977.

Chickens congenitally infected with an avian leukosis virus (ALV) of antigenic subgroup A and normal chickens were challenged with subgroups B and C of Rous sarcoma virus (RSV) and tumor induction and growth as well as humoral antibody to viral envelope antigen (VEA) and tumor-specific surface antigen (TSSA) were measured. Groups of 3-wk-old control or ALV F42 congenitally infected birds were challenged with 10, 100, or 1,000 focus-forming units of the Schmidt-Ruppin or Prague strains of RSV. The presence of either anti-TSSA or anti-VEA antibody did not reflect the tumor status of the host, and there were no significant differences in the incidence of anti-VEA or anti-TSSA antibodies in the birds in either group. Tumors in leukosis virus-infected birds grew progressively, but tumors in the RSV-challenged normal birds regressed. The growth rate and size of the tumors were higher in congenitally infected birds, but there was no influence of congenital leukosis virus infection on the RSV latent period or tumor incidence. Congenital infection of the chicken with a leukosis virus alters solid tumor growth to the detriment of the host. (41 refs.)

77-2017 **Presence of Ribonucleotide Sequences Complementary to Rous Sarcoma Virus (RSV) RNA in Chicken Cells Infected with RSV.** (Eng.) Shaposhnikov, J. D. (Lab. Biochemistry, Petrov Res. Inst. Oncology, 68 Leningradskaya St., Pesochny-2, Leningrad 188646, USSR) Ratovitski, E. A.; Kuznetsov, O. K. *Cancer Lett* 2(6): 349-354; 1977.

The homology between the RNA's from chicken Rous sarcoma cells and Rous sarcoma virus (RSV) virions was examined. RNA was isolated from purified RSV virions and from the nuclei, mitochondria, and free and membrane-bound polyribosomes of Rous sarcoma cells. Preliminary results of the hybridization between RSV virion <sup>125</sup>I-RNA and unlabeled RNA from the various subcellular fractions of Rous sarcoma cells showed that RNAase-resistant hybrids were formed only with the RNA from membrane-bound polyribo-



somes and mitochondria. Mitochondrial RNA contained approx 12% ribonucleotide sequences complementary to those of virion RNA. The mitochondrial RNA of the normal chicken embryo and normal mouse liver contained 2.2% and 1.8% complementary regions, respectively. Rous sarcoma membrane-bound polyribosome RNA contained 4.5% complementary sequences, whereas the normal chicken embryo and mouse liver membrane-bound polyribosome RNA's contained only 3.3% and 0.9% complementary sequences. The complementary regions in the RNA's from these subcellular fractions may be due to the presence of RNA-synthesizing activity in oncornavirus virions and to the presence of oncornavirus-like particles in tumor mitochondria. (18 refs.)

- 77-2018 A Mutant of Rous Sarcoma Virus with a Conditional Defect in the Determinant(s) of Viral Host Range.** (Eng.) Mason, W. S. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA. 19111) Yeater, C. *Virol* 77(2): 443-456; 1977.

A mutant of the Prague strain of Rous sarcoma virus of subgroup C with a conditional temperature-sensitive defect in the viral host range of subgroup C and defective in the production of virus envelope glycoproteins (labelled G35 and G85) was isolated and characterized. The temperature-sensitive mutant, tsPH734PR-C, was isolated from wtPR-C parents grown in the presence of 5-azacytidine. The permissive temperature for the mutant is 35 C and the nonpermissive temperature is 41 C. TsPH734-C forms foci equally well at 41 C as does the wtPR-C but produces progeny 100 times less effectively at 41 C. Tests also showed that tsPH734-C is not complemented for replication by coinfection with avian leukosis virus (RAV-6), subgroup B viruses, or the defective Bryan strain of Rous sarcoma virus BH-RSV(-). It was also shown via gel electrophoresis that tsPH734-C is defective in the utilization and synthesis of virus envelope glycoproteins G35 and G85. It is concluded that there is a mutation in the virus gene(s) that code(s) for these proteins. (33 refs.)

- 77-2019 Formation of Reticuloendotheliosis Virus Pseudotypes of Rous Sarcoma Virus.** (Eng.) Sawyer, R. C. (Rockefeller Univ., New York, NY 10027) Hanafusa, H. *J Virol* 22(3): 634-639; 1977.

Biological interactions between the defective Bryan strain of Rous sarcoma virus (BH-RSV), which is unable to direct the synthesis of its envelope glycoprotein, and two different reticuloendotheliosis viruses were studied. Superinfection of chicken embryo fibroblasts transformed by BH-RSV with  $1 \times 10^6$  infectious units of reticuloendotheliosis virus strain T (REV-T) or spleen necrosis virus (SNV) resulted in the production of infectious sarcoma virus pseudotypes. These pseudotypes were neutralized by antiserum prepared against SNV and were unable to infect chicken cells preinfected with either REV-T or SNV. The results suggest that BH-RSV is able to use the glycoprotein from REV to form infectious

pseudotypes. On the other hand, neither REV-T nor SNV was able to supply a functional reverse transcriptase to the polymerase-negative mutant BH-RSV $\alpha$ , nor was REV-T or SNV able to complement the defect in the internal protein gene of the temperature-sensitive avian sarcoma virus mutant NY45. (29 refs.)

- 77-2020 The Role of the Subependymal Plate in Avian Sarcoma Virus Brain Tumor Induction: Comparison of Incipient Tumors in Neonatal and Adult Rats.** (Eng.) Copeland, D. D. (Dept. Pathology, Duke Univ. Medical Center, Durham, NC 27710) Bigner, D. D. *Acta Neuropathol (Berl)* 38(1): 1-6; 1977.

The role of the subependymal cell plate in the genesis of an avian sarcoma virus (ASV)-induced brain tumors in F-344 rats was evaluated. A comparison of the relationship of incipient tumors in adult and neonatal rats to the subependymal cell region was also made. Rats were inoculated at 1 or 133 days of age with  $8.7 \times 10^4$  focus-forming units of B-77-ASV. Among rats inoculated on the first postnatal day, the first microtumor was found in the subependymal region of a rat killed 12 days after treatment. Microtumors were discovered in all neonatal rats killed in the fifth week after inoculation. Incipient tumors were typically found in the subependymal region of the lateral ventricles. Among rats inoculated at 133 days of age, only 1 incipient tumor was found in 10 rats killed 32 days after treatment. By 90 days after inoculation, the proportion of adult rats with tumors increased to 64%. Only one incipient tumor was located in the subependymal region; the remainder were found in the grey and white matter of the cerebral cortex. The role of the subependymal plate is discussed. (21 refs.)

- 77-2021 Analysis of Precursors to the Envelope Glycoproteins of Avian RNA Tumor Viruses in Chicken and Quail Cells.** (Eng.) Moelling, K. (Max-Planck-Institut für Molekulare Genetik, D-1000 Berlin, W. Germany) Hayami, M. *J Virol* 22(3): 598-607; 1977.

The intracellular synthesis of glycoproteins gp85 and gp37 of the B77 strain of Rous sarcoma virus was studied by immune precipitation with monospecific antiserum. Labeled gp85 and gp37 were detected from lysates of B77-transformed cells after long-term labeling with radioactive glucosamine or phenylalanine. Immune precipitates from lysates of cells pulse-labeled for a short time revealed a glycoprotein of 92,000 molecular wt (gp92). This precursor was stable in B77-transformed Japanese quail cells for several hours, but in chicken cells it could be chased within a few hours into glycoproteins gp85 and gp37. Similarly, the precursor for the structural viral proteins, pr76, persisted much longer in quail cells than in chicken cells. During very short pulses or in the presence of a glucosamine block (25 mM glucosamine), the antiserum against the viral envelope glycoprotein detected a precursor (p70) of higher electrophoretic

mobility of approx 70,000 molecular wt. Fucose label entered gp92 and gp85 as well as p70. Trypsin treatment of virion-bound gp85 in vitro generated two discrete glycoproteins of 62,000 and 45,000 molecular wt, but did not result in an increase in the amount of gp37. (32 refs.)

**77-2022 Sequence Studies of the Terminal Regions of AMV RNA: Possible Redundancy (Meeting Abstract).** (Eng.) Stoll, E. (Institut für Molekularbiologie I, Universität Zürich, CH-8093 Zürich, Switzerland) Billeter, M. A.; Palmenberg, A.; Weissmann, C. *Experientia* 33(6): 801; 1977. (no refs.)

**77-2023 Oligodeoxyribonucleotides Primed In Vitro Reverse Transcriptase of Various RNAs (Meeting Abstract).** (Eng.) Bromley, P. A. (Département de Biologie Moléculaire, Université de Genève, CH-1211 Genève, Switzerland) Darlix, J. L.; Spahr, P. F. *Experientia* 33(6): 813; 1977. (no refs.)

**77-2024 Reversible Inhibition of Interferon-induced Antiviral State by Deoxyadenosine.** (Eng.) Matsuno, T. (Dept. Measles Virus, National Inst. Health Japan, Musashimurayama, Tokyo 190-12, Japan) Shirasawa, N.; Kohno, S. *Acta Virol* 20(6): 466-471; 1977.

A study was made of the effects of certain nucleotides upon the development of the antiviral state (AVS) induced by interferon in chick embryo fibroblasts. The concentrations of adenosine, adenosine, deoxyadenosine, and 3'-deoxyadenosine (cordycepin) required to bring about a 50% reduction in the AVS were 4.6, 0.7, 1.0, and 0.015 mM, respectively. The inhibitory effects were reversible. Actinomycin D also inhibited the development of the AVS. The inhibition appeared to be related to an inhibition of RNA synthesis, especially nucleolar RNA synthesis. (10 refs.)

**77-2025 Murine Xenotropic Type C Viruses. III. Phenotypic Mixing with Avian Leukosis and Sarcoma Viruses.** (Eng.) Levy, J. A. (Cancer Res. Inst., Univ. California Medical Center, San Francisco, CA 94143) *Virology* 77(2): 811-825; 1977.

Phenotypic mixing between murine and avian C-type viruses was investigated in chicken, duck, and mouse embryo fibroblasts. Pseudotypes of Rous sarcoma virus (RSV) were obtained that carry envelope determinants derived from xenotropic or ecotropic murine leukemia virus (MuLV). Mouse sarcoma virus (MSV) pseudotypes with the envelope characteristics of subgroup C and E avian leukosis viruses were obtained. RSV pseudotypes produced with MuLV could in-

fect mammalian cells, including human cells; MSV pseudotypes with avian leukosis virus infected chicken cells. There was some suppression of transforming activity in some of the heterologous hosts. Exogenous infection of mammalian cells by RSV(MuLV) resulted in the efficient production of infectious progeny virus as well as in the transformation of the heterologous host. The permissiveness of the host cell for MuLV replication is important to the system, as indicated by the host range of the progeny virus. These results demonstrate that sarcoma genomes can be passed easily to heterologous cells, including those of a different taxonomic class of host animal. (44 refs.)

**77-2026 Murine Xenotropic Type C Viruses. II. Phenotypic Mixing with Mouse and Rat Ecotropic Type C Viruses.** (Eng.) Levy, J. A. (Cancer Res. Inst., Univ. California Medical Center, San Francisco, CA 94143) *Virology* 77(2): 797-810; 1977.

Interference patterns for murine xenotropic (X-tropic) and ecotropic C-type viruses and the phenotypic mixing that results from coinfection of mouse or rat cells with these two different murine C-type virus classes were investigated. Phenotypic mixing occurred readily between the murine X-tropic and ecotropic C-type viruses. There was also no interference between mouse and rat endogenous C-type viruses, and they could undergo phenotypic alterations. The absence of virus interference permits the replication of X-tropic and ecotropic murine leukemia virus in the same cell, which results in phenotypic alterations involving the antigenic coat and host range of the progeny viruses. Phenotypic mixing may occur as early as 1 hr after infection, and it enhances the potential for the spread of oncogenic genomes among all animal species, including man. These events may play a role in evolution, possibly by inducing somatic cell changes in addition to transformation. Selective intracellular regulating systems for C-type viruses were demonstrated. X-tropic virus replicated less efficiently than ecotropic virus in mouse cells. Ecotropic virus replication was diminished in heterologous cells compared to that of X-tropic virus. The results suggest that the predominant block in mouse cells to X-tropic viruses is at the surface or penetration level and that incoming and integrated viral genes respond to different kinds of host cell control. (55 refs.)

**77-2027 Type C Oncornaviruses in a Capuchin Monkey (*Cebus Apella*) Previously Infected with *Schistosoma Haematobium*.** (Eng.) Kalter, S. S. (Southwest Foundation for Res. and Education, PO Box 28147, San Antonio, TX 78284) Kuntz, R. E.; Heberling, R. L.; Smith, G. C.; Moore, J. A.; McCullough, B. *Lab Anim Sci* 27(1): 122-124; 1977.

Electron microscopy revealed C-type virus particles in the lymph nodes of a capuchin monkey infected 3.5 yr earlier



with *Schistosoma haematobium*. No viral particles were detected in a bladder tumor (papillary carcinoma) that developed 25 mo after exposure to *S. haematobium* and later regressed completely. (16 refs.)

- 77-2028 Subunit Interactions in Polyoma Virus Structure.** (Eng.) Etchison, D. (Dept. Microbiology, Medical Center, Univ. Utah, Salt Lake City, UT 84132) Walter, G. *Virology* 77(2): 783-796; 1977.

Purified polyoma virions were treated with increasing concentrations of sodium dodecyl sulfate (SDS, 0.02%-0.06%), isolated from sucrose gradients, and separated by polyacrylamide gel electrophoresis (PAGE) to yield subunits in the following order: VP3, VP2, and VP1. The final structure contained disulfide-cross-linked VP1, and it resembled empty capsids by electron microscopy. This structure had the same hemagglutinating activity as intact virions isolated from the same sucrose gradient. The results indicate that the hemagglutinin is a disulfide-linked polymer of VP1. Disulfide-cross-linked and uncross-linked VP1 could be distinguished in polyoma-infected cell lysates by PAGE. The DNA of the majority of the virions was released as soon as a few proteins dissociated. With very low concentrations of SDS, however, an 80S structure that contained DNA but that lost a few minor proteins was generated. Most of the 80S particles could be converted to empty capsids by slightly higher concentrations of SDS, but a residual portion retained DNA even after treatment with high concentrations of salt or with concentrations of SDS at which all minor proteins were removed. This SDS-induced 80S structure is probably an intermediate in virus denaturation rather than a complex formed between free DNA and empty capsids. (21 refs.)

- 77-2029 Multiple Free Viral DNA Copies in Polyoma Virus-transformed Mouse Cells Surviving Productive Infection.** (Eng.) Magnusson, G. (Dept. Biochemistry, Stanford Univ., Stanford, CA 94305) Nilsson, M. G. *J Virol* 22(3): 646-653; 1977.

The properties of clonal isolates of mouse 3T6 cells surviving infection with polyoma virus at a multiplicity of 50 plaque-forming units/cell were investigated, particularly the amounts and characteristics of viral DNA isolated from these cells. The clones were resistant to a second infection, but in some cases viral functions could be at least partially expressed during reinfection, as indicated by an increase in the number of tumor (T)-antigen-positive cells. One clone in which T antigen was stimulated was investigated in detail. These cells were transformed, and they produced low amounts of virus. The resistance of these cells to reinfection may be explained by interference from the viral DNA in the cells. This DNA had the normal physical characteristics of polyoma DNA, but electrophoresis of linearized molecules showed that it was slightly larger than wild-type polyoma DNA. Mapping with restriction endonucleases revealed that the addition was

about 5% of the wild-type genome and that it was located close to the origin of DNA replication. This DNA had a 10-fold lower infectivity than wild-type polyoma DNA. It is not known why some of the cells survived the initial infection but the most likely explanation is that the original cultures contained cells that had a decreased susceptibility to polyoma virus. (24 refs.)

- 77-2030 Properties of the Polyoma Viruses Induced from BHK-21 Cells Transformed by A Gene Mutants.** (Eng.) Anderson, D. M. (Dept. Biological Chemistry, Univ. Michigan, Ann Arbor, MI 48109) Folk, W. R. *J Virol* 22(3): 826-829; 1977.

The properties of the polyoma viruses induced from hamster BHK-21 cells transformed by polyoma A gene mutants were investigated and compared with those of the viruses originally used for transformation. Several procedures were used to generate stocks of infectious virus from the inducible transformed BHK-21 cells. Viruses present in these stocks hemagglutinated guinea pig RBC with the same efficiency as the parental virus, and they displayed thermosensitive plaque-forming ability on whole mouse embryo cells. The viruses from the induced cells also shared considerable, if not complete, sequence complementarity with polyoma DNA. The persistence of the transformed phenotype (growth in soft agar) in these polyoma A gene-transformed cells at a temperature at which initiation of rounds of viral DNA replication is blocked (in mouse cells) and at which there is no detectable autonomous viral DNA replication indicates that the activity required to maintain the transformed phenotype is functionally separable from the A gene activity required for the initiation of autonomous viral DNA replication. (10 refs.)

- 77-2031 Isolation and Characterization of the Replicative Intermediates and Progeny DNA of the Provirus Kilham Rat Virus (KRV)** (Meeting Abstract). (Eng.) Li, A. T. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Lavelle, G. C.; Tennant, R. W. *Fed Proc* 36(3): 1085; 1977. (no refs.)

- 77-2032 Sequence Homology Between the Structural Polypeptides of Minute Virus of Mice.** (Eng.) Tattersall, P. (Dept. Cell Biology, Roche Inst. Molecular Biology, Nutley, NJ 07110) Shatkin, A.; Ward, D. C. *J Mol Biol* 111(4): 375-394; 1977.

The primary sequences of three polypeptide species found in DNA-containing particles of minute virus of mice were compared by proteolytic fingerprinting of their radioiodinated derivatives using trypsin and  $\alpha$ -chymotrypsin. The three species are A (Mr = 83,000), B (Mr = 64,000), and C (Mr =

61,000). The sequences for B and C were almost identical, supporting the idea that these two proteins are precursor and product, respectively. The occurrence of C-specific spots in both types of fingerprints suggests that cleavage *in vivo* is performed by a protease with unique specificity. The entire sequence of B appears to be present in A. In addition to the 36 iodopeptides similar to both, 9 A-specific peptides that contain a preponderance of arginyl and lysyl residues were observed. Both the A polypeptide in either a full or empty virion and the B polypeptide in empty particles were resistant to tryptic or chymotryptic digestion *in vitro*. The *in vivo* observation that empty virions contain both A and B but not C correlates well with this result. A scheme for the maturation of the papovavirus virion is proposed. *In vivo* B-C cleavage appears to decrease the receptor-binding activity of the particle, whereas reversal of this conformational change restores full infectivity and hemagglutinating activity. (34 refs.)

**77-2033** Primer Function of Proline tRNAs in the Reverse Transcription of Murine Leukemia Virus 35S RNA (Meeting Abstract). (Eng.) Yang, W. K. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) *Fed Proc* 36(3): 847; 1977. (no refs.)

**77-2034** Characterization of the Moloney Leukemia Virus-Determined Cell Surface Antigen and Its Relation to the Known Murine Leukemia Virion Proteins (Meeting Abstract). (Eng.) Siegert, W. (Universitätsklinikum Grosshadern, Medizinische Klinik II, Marchioninstr. 15, D-8000 Munich 70, W. Germany) Fenyo, E. M.; Klein, G. *Hoppe Seylers Z Physiol Chem* 358(3): 308; 1977. (no refs.)

**77-2035** Donation of N- or B-tropic Phenotype to NB-tropic Murine Leukemia Virus During Mixed Infections. (Eng.) Kashmiri, S. V. (Litton Bionetics, Inc., Bethesda, MD 20014) Rein, A.; Bassin, R. H.; Gerwin, B. I.; Gisselbrecht, S. *J Virol* 22(3): 626-635; 1977.

The IC isolate of Moloney murine leukemia virus (MuLV), which is NB-tropic, was grown in 3T3FL cells producing conditionally defective or defective virus particles derived from N- or B-tropic MuLV. The infectious MuLV released by cells infected with NB- and B-tropic viruses was sensitive to Fv-1n restriction, but produced NB-tropic progeny upon passage. Similarly, the virus particles produced by cells dually injected with NB- and N-tropic viruses were sensitive to Fv-1b restriction, but gave rise to NB-tropic progeny. These results indicate that the NB-tropic IC isolate can acquire sensitivity to Fv-1 restriction by phenotypic mixing with N- or B-tropic MuLV. NB-tropic MuLV may be normally insensitive to Fv-1 restriction because it does not synthesize the determinants of tropism. (36 refs.)

**77-2036** Biochemical and Immunological Characterization of the Major Envelope Glycoprotein gp69/71 and Degradation Fragments from Rauscher Leukemia Virus. (Eng.) Krantz, M. J. (Montreal General Hosp. of Cancer Inst., Montreal, Canada) Strand, M.; August, J. T. *J Virol* 22(3): 804-815; 1977.

The glycoproteins of Rauscher murine leukemia virus, propagated in a BALB/c mouse bone marrow cell line, were characterized by immunoprecipitation with monospecific antiserum and tryptic peptide analysis. Antiserum to the purified Rauscher envelope glycoprotein of approx 69,000 and 71,000 daltons (gp69/71) reacted as well with a number of other components of several murine oncornaviruses (Friend, Moloney, Gross, and BALB xenotropic virus 2) of approx 45,000, 32,000, and 15,000 daltons. Polypeptides of similar size were also produced by limited proteolysis of purified gp69/71; these degradation fragments contained carbohydrate, as shown by the incorporation of  $^3\text{H}$  from sodium borohydride after neuraminidase and galactose oxidase treatment. Tryptic peptide analysis of glycoproteins separated by polyacrylamide gel electrophoresis indicated that the 69,000- and 71,000-dalton virion components were nearly identical, as were the primary degradation fragments of 45,000 and 32,000 daltons. Analysis of the immunologic properties of the glycoproteins demonstrated that the 71,000-, 69,000-, and 32,000-dalton glycoprotein shared inter-species and group-specific antigenic determinants. In contrast, the 45,000-dalton glycoprotein lacked detectable interspecies and some of the group-specific reactivity. It is concluded that the four viral glycoproteins examined (71,000, 69,000, 45,000 and 32,000 daltons) are derived from the same viral gene. (45 refs.)

**77-2037** Partial Characterization of a P70 Proteolytic Factor that is Present in Purified Virions of Rauscher Leukemia Virus (RLV). (Eng.) Yoshinaka, Y. (Worcester Foundation Experimental Biology, Shrewsbury, MA) *Biochem Biophys Res Commun* 76(1): 54-63; 1977.

A p70 proteolytic factor present in purified virions of Rauscher leukemia virus (RLV) was isolated and partially characterized. The proteolytic factor was induced by incubation of RLV at 22 C in the presence of Nonidet P40 (NP40). Only p70 was cleaved at low detergent concentrations (0.1%-0.3%) when RLV was incubated for 16 hr. A similar cleavage of p70 only occurred at short times (1-1.5 hr) when the NP40 concentration was higher (2%). These cleavages were accompanied by an increase in a 40- to 42-dalton protein (p40-42). At higher concentrations or longer times, p40-42 was also cleaved. This indicates that the cleavage occurs in two steps, first to p40-42 and then to p30. The *in vitro* incubation of labeled immature core subparticles enriched in p70 with a partially purified proteolytic factor fraction showed an enrichment in both p40-42 and p30. This activity was inhibited



by tosylsulfonyllysyl chloromethyl ketone (1-10 mM) but not by tosylsulfonylphenylalanyl chloromethyl ketone or carbobenzyloxyphenylalanyl chloromethyl ketone ( $\geq 2$  mM), suggesting that the factor is more trypsin- than chymotrypsinlike. These findings coupled with previously published results indicate that several different proteolytic activities are involved with murine leukemia virus assembly at different times, suggesting a host rather than viral origin for all of them. (18 refs.)

- 77-2038 Biological and Structural Pleomorphism of the Oncornavirus Envelope Glycoprotein, gp70.** (Eng.) Del Villano, B. C. (Dept. Immunopathology, Scripps Clinic and Res. Foundation, La Jolla, CA 92037) Kennel, S. J.; Lerner, R. A. *Contemp Top Immunobiol* 6: 195-207; 1977.

Studies using SCRF 60 A cells, a lymphoblastoid cell line derived from an NZB (New Zealand black) mouse, showed that the murine leukemia virus (MuLV) gp70 is a component of the surfaces of virus particles and that it is also found in the cell membranes of infected cells, even in regions where there are no virus particles. In another study, 78% of the MuLV-induced lymphomas had detectable gp70. This study also indicated that gp70 may be structurally pleomorphic. The molecular wt of the gp70 from the thymus lymphoma cells of a single BALB/c mouse was 75,000 and that of the spleen lymphoma cells of the same mouse was 69,000. Gp70 was also found in most normal mouse tissues, and its molecular wt varied according to its tissue of origin. Relatively large amounts of gp70 were associated with the surface of cells from mice that were positive for the GIX differentiation antigen, but little or no gp70 was found on cells in the GIX-strains. These findings indicate that proteins related to oncornavirus gene products exhibit both biological pleomorphism (in that gp70 may be a component of viruses and normal and malignant cells) and structural pleomorphism (in that the relative size of the gp70 from different mouse tissues is characteristic of the tissue of origin). (27 refs.)

- 77-2039 Virus Protein p30 in Blood Predicts Development of Leukaemia in Mice Injected with MuLV.** (Eng.) Ulrich, K. (Fibiger-Lab., DK Copenhagen O, Denmark) Nexø, B. A. *Nature* 267(5613): 723-724; 1977.

Thirteen BALB/c mice were inoculated at birth with D2BAL-10 murine leukemia virus (MuLV: 50  $\mu$ l sc), which was isolated from a dimethyl(a)benzanthracene-induced DBA/2 leukemia. There was found to be a significant correlation between high blood concentrations of the virus core protein p30 early in life and the subsequent development of leukemia. Eight mice showed a significant increase in p30 levels (760-1,200 nanograms/ml) within 36 days; 6/8 developed leukemia (within 247 days), as did 2/4 mice (within 310-380 days) with high p30 concentrations first measured at 200 days of age. Twelve of 13 controls were alive without signs of disease after 13 mo. (8 refs.)

- 77-2040 Simultaneous Chemical Induction of MTV and MLV In Vitro.** (Eng.) Links, J. (Netherlands Cancer Inst., Div. Virology, Sarphatistraat 108, Amsterdam Netherlands) Calafat, J.; Buijs, F.; Tol, O. *Eur J Cancer* 13(6): 377-387; 1977.

Latent endogenous mammary tumor virus (MTV) and latent endogenous mouse leukemia virus (MLV) were simultaneously chemically induced with a concomitant cell morphological alteration in tissue-cultured baby mouse kidney cell (BMKC) derived from the low mammary tumor mouse strains BALB/c (substrains He.A and Crgl.A) and C57BL/Li.A. 3-Methylcholanthrene was used with BMKC prepared from BALB/c/He.A and BALB/c/Crgl.A mice, and 5-bromodeoxyuridine (30  $\mu$ g/ml) was used with BMKC prepared from C57BL/Li.A. Aggregates (pocks) of small rounded epithelioid cells appeared on the BMKC monolayer several weeks after chemical treatment. Cell lines produced from single pocks released MTV (B-particles) and MLV (C-particles) as found by electron microscopy and reverse transcriptase determinations. All isolated cell lines grew well when serum was omitted from the medium. In female syngeneic BALB/c/He.A mice, MC-altered BMKC induced early (< 300 days) mammary tumor and early (< 200 days) leukemia. A cell-free extract from these chemically altered BMKC induced similar early tumors. Untreated control BMKC cultures did not show the described alterations and properties. The data support the conclusion that MTV can be present in "virus-free" mouse cells from which the virus can be chemically induced simultaneously and in the same way as MLV. (43 refs.)

- 77-2041 Biological Activities of Murine Mammary Tumour Virus In Vitro: Increased Macromolecular Syntheses in Mouse and Hamster Kidney Cells; Production of B- and C-Particles in the Mouse Cells.** (Eng.) Links, J. (Div. Virology, Netherlands Cancer Inst., Sarphatistraat 108 Amsterdam, Netherlands) Tol, O.; Calafat, J.; Buijs, F. *Eur J Cancer* 13(6): 539-548; 1977.

Murine mammary tumor virus (MTV) induced an increased net synthesis of DNA, RNA, and protein in short-term cultures of baby mouse kidney cells (BMKC) from two different BALB/c mouse substrains (He.A and Crgl.A). The dose-response ratio went through a max, but growth inhibition was not observed. Similar effects were induced in baby hamster kidney cells (BHK 21/C 13). In long-term cultures of MTV-infected BMKC, foci of piled-up, small, round epithelioid cells appeared on the monolayers. Cell lines derived from these foci grew well in media without serum and produced biologically active MTV (B particles) and murine leukemia virus (MLV; C particles). Higher in vitro passages of these cell lines induced fewer early (< 300 days) mammary tumors and more cases of leukemia than the lower passages after ip injection. Cell-free extracts induced similar tumors. Control BMKC did not acquire these properties. The possibility that the induced MTV and MLV were of endogenous origin is

discussed. Induction of endogenous MLV by MTV could play a role in the spontaneous in vivo transformation of murine mammary adenocarcinoma into sarcoma. (57 refs.)

77-2042 **Electrophoretic Analysis of the Molecular Weight of Murine Mammary Tumor Virus RNA.** (Eng.) Dion, A. S. (Inst. Medical Res., Camden, NJ 08103) Heine, U. I.; Pomenti, A. A.; Korb, J.; Weber, G. H. *J Virol* 22(3): 822-825; 1977.

The molecular wt of native and subunit RNA's of murine mammary tumor virus (MuMTV) was determined by polyacrylamide gel electrophoresis and compared with that of well-characterized avian cellular RNA's and tobacco mosaic virus RNA. From extrapolations of semilog plots of the molecular wt of the standard RNA's versus relative electrophoretic mobilities and Ferguson plots, the subunit and native RNA's of MuMTV were found to have molecular wts of  $2.93 \times 10^6$  and  $6.45 \times 10^6$ , respectively. These data indicate a dimeric structure for the native RNA, which has also been observed for other viral RNA's. This structure is similar in conformation to the subunit RNA. (15 refs.)

77-2043 **In Vitro Translation of Harvey Murine Sarcoma Virus RNA.** (Eng.) Parks, W. P. (Lab. Tumor Virus Genetics, NCI, Bethesda, MD 20014) Scolnick, E. M. *J Virol* 22(3): 711-719; 1977.

The viral RNA of Harvey murine sarcoma virus (Ha-SV), which does not encode for any known viral structural polypeptides, was translated in a nuclease-digested, cell-free system. The major protein product of the in vitro translation reaction has a molecular wt of 21,000 and is initiated faithfully with  $^{35}\text{S}$ -formylmethionine from formyl- $^{35}\text{S}$ -methionyl-transfer RNA-F-MET. The p21 polypeptide was clearly distinct from the products of reactions translated in parallel experiments from the RNA of the Moloney strain of C-type helper virus used to pseudotype the Ha-SV. The intensity of p21 on polyacrylamide slab gels was in direct proportion to the concentration of Ha-SV RNA in different viral RNA preparations. The p21 polypeptide represents the first protein marker for Ha-SV, which was originally isolated by passaging Moloney leukemia virus in a Chester Beatty rat. The possibility that the protein is the product of the rat portion of the Ha-SV genome is discussed. (37 refs.)

77-2044 **Different Mechanisms for Morphologic Reversion of a Clonal Population of Murine Sarcoma Virus-transformed Nonproducer Cells.** (Eng.) Bensinger, W. I. (Lab. RNA Tumor Viruses, NCI, Bethesda, MD 20014) Robbins, K. C.; Greenberger, J. S.; Aaronson, S. A. *Virology* 77(2): 750-761; 1977.

Several morphologic revertants were isolated from a clonal line of BALB/3T3 cells nonproductively transformed by Kirsten mouse sarcoma virus (KiMSV). Enrichment for these revertants was obtained by selective kill of cells expressing the transformed phenotype in semisolid methylcellulose medium. Each revertant clone was obtained from a separate mutagenized (mitomycin C or 8-azaguanine) cell culture. The newly isolated revertants were indistinguishable from the BALB/3T3 cells in morphology and in vitro growth properties. One group of revertants, which contained reversibly altered sarcoma viral genomes, expressed high levels of sarcoma viral RNA. Wild-type transforming virus became rescuable at a time when cells in the population spontaneously retransformed (1 in  $10^6$ - $10^8$  cell generations). There was no evidence of complementation or recombination between the altered sarcoma viruses present in these revertant lines. A second class of revertants was very stable to retransformation and never demonstrated rescuable sarcoma virus. Analysis of KiMSV (Ki murine leukemia virus) [KiMSV(KiMuLV)] DNA and RNA in these cells indicated that they had lost sarcoma virus information. These results indicate that both alteration and loss of sarcoma virus information are reproducible causes of reversion to the nontransformed state in this system. (32 refs.)

77-2045 **Characterization of Visna Virus Envelope Neuraminic Acid.** (Eng.) August, M. J. (Virology Lab./151, Veterans Admin. Hosp., West Haven, CT 06516) Harter, D. H.; Compans, R. W. *J Virol* 22(3): 832-834; 1977.

The ability of visna virus to inhibit influenza virus hemagglutination and the biological function of its envelope neuraminic acid were examined. The binding of visna virus to the surface of purified WSN strain influenza virions prevented the characteristic influenza hemagglutination. Pretreatment of visna virions with neuraminidase, however, completely abolished this hemagglutination inhibition (HI) activity. The presence of neuraminic acid in visna virions was demonstrated by direct assay, in which  $0.39 \mu\text{g}$  of neuraminic acid was detected per  $100 \mu\text{g}$  of virion protein. Neuraminidase treatment of visna virus did not significantly affect attachment, infectivity, or virus-induced cell fusion in sheep choroid plexus cell monolayers. The retention of full biological activities of neuraminidase-treated visna virus is in contrast to the results obtained with vesicular stomatitis virus, indicating that the biological function of envelope sialic acid varies with different enveloped viruses. (14 refs.)

77-2046 **Virus-related Genetic Material in Cancer and Blood Specimens from Humans (Letter to Editor).** (Eng.) Arida, E. (Wenner-Gren Inst., Stockholm Univ., Stockholm, Sweden) Hultin, T.; Emas, S.; Ware, J. *JAMA* 237(26): 2812-2813; 1977.



Virus-related genetic material was recently detected in tumor and blood specimens of three patients with colon cancer and five patients with rectal cancer. Three of the patients had liver metastases and one had peritoneal metastases. The technique was similar to one used to detect viral genes in murine sarcoma-transformed cells. (5 refs.)

- 77-2047 Characterization of Human Papovavirus RFV: Use of Iodinated Viral DNA to Detect Viral DNA Sequences in Cellular DNA.** (Eng.) Miao, R. (Dept. Biomedical Res., Stanford Res. Inst., 333 Ravenswood Ave., Menlo Park, CA 94025) Dougherty, R. M. *Virology* 77(2): 856-859; 1977.

The fraction of the viral genome present in a transformed cell line (RF-194) derived from hamster embryo fibroblasts infected with a new human papovavirus (RFV, isolated from the urine of a renal transplant patient) was determined using saturation reassociation of cellular DNA and  $^{125}\text{I}$ -labeled RFV DNA. Only 40% of the viral genome was present in the transformed cells, indicating that only a fraction of the viral genome is necessary to maintain the transformed state. This same fraction was sufficient to maintain the tumorigenicity of RF-194 cells. The high specific activity of  $^{125}\text{I}$ -RFV DNA permitted the quantitative determination of RFV sequences using  $< 260 \mu\text{g}$  of cellular DNA. It should be possible to quantitate and identify viral sequences in tumors induced in vivo in animals using these techniques. (9 refs.)

- 77-2048 Rise in Antibodies to Human Papova Virus BK and Clinical Disease (Letter to Editor).** (Eng.) Noordaa, J. V. (Virus Lab., University Amsterdam, Amsterdam, The Netherlands) Wertheim-van Dillen, P. *Br Med J* 1(6014): 1471; 1977.

A retrospective study of serial serum samples from 77 patients revealed three cases of primary BK virus infection, indicated by the appearance of hemagglutination-inhibiting antibodies. Acute upper respiratory symptoms accompanied by neurological involvement occurred in all three patients, a 5-yr-old child and adults aged 33 and 45 yr. The adults also developed acute inflammatory polyradiculoneuropathy. (3 refs.)

- 77-2049 Detection of Mumps Virus Antigens in Hodgkin's Disease Tissues.** (Eng.) Truant, A. L. (Dept. Microbiology and Immunology, Univ. Oregon Health Sciences Center, Portland, OR 97201) Hallum, J. V. *Oncology* 33(5/6): 241-245; 1976.

Mumps virus antigens were found by indirect immunofluorescence in the biopsied tissues from 12 Hodgkin's disease (HD) patients, but not in the lymph node tissue of 4 non-HD lymphoma patients. Impression smears from 2 spleen and 10 lymph node HD specimens revealed viral antigens in the cytoplasm and/or nucleus. A measles virus screening of a portion of the tissues revealed that 6/7 HD tissues demonstrated measles virus-specific immunofluorescence. Six HD lymph nodes were measles virus-positive, but one HD spleen was measles virus-negative, although all were mumps virus-positive. In control tissues, measles antigens were detected in 9/18 tissues and mumps antigens in 7/31. All tissues tested were negative for the presence of Newcastle disease virus antigens by direct immunofluorescence. (25 refs.)

- 77-2050 Epstein-Barr Virus Antibodies in Tonsillar Carcinoma Patients.** (Eng.) Vonka, V. (Dept. Experimental Virology, Inst. Sera and Vaccines, Prague, Czechoslovakia) Sibl, O.; Suchankova, A.; Simonova, I.; Zavadova, H. *Int J Cancer* 19(4): 456-459; 1977.

The presence of Epstein-Barr (EB) virus antibodies in a group of Czechoslovak tonsillar cancer (TC) patients is reported. Sera were obtained from 10 patients with active TC, 8 patients who were in remission, and from 18 sex- and age-matched controls. Serological tests for viral capsid antigen (VCA), early antigen (EA), and nuclear antigen (EBNA) were conducted. All the TC patients possessed VCA antibodies in titers ranging from 1:20 to  $> 1:640$ . Antibodies to EA were discovered in 14 patients, and in 5 the titers were at least 1:160. EBNA antibodies were found in the sera of all patients. The geometric mean titers in the patients with active TC were no different from those of the patients in remission. In all three tests, antibodies were detected more frequently and in significantly higher titers in the patients than in the controls. (17 refs.)

- 77-2051 Presence of Epstein-Barr Virus Receptors, but Absence of Virus Penetration, in Cells of an Epstein-Barr Virus Genome-Negative Human Lymphoblastoid T Line (Molt 4).** (Eng.) Menezes, J. (Dept. Microbiology and Immunology, Univ. Montreal, Ste-Justine Hosp., Montreal H3T 1C5, Quebec, Canada) Seigneurin, J. M.; Patel, P.; Bourkas, A.; Lenoir, G. *J Virol* 22(3): 816-821; 1977.

Interactions between Epstein-Barr virus (EBV) and an EBV receptor-positive, genome-negative human lymphoid T cell line (Molt 4) were investigated at three different levels: (1) virus receptors by membrane immunofluorescence using two different EBV strains; (2) virus attachment to, and penetration in, the cell by electron microscopy (EM); and (3) the expression of EBV-induced antigens in infected cells. The binding of EBV to the cell surface was demonstrated by mem-

brane immunofluorescence and EM. No detectable EBV-induced intracellular antigen was found by immunofluorescence. This finding is supported by the EM observation that EBV particles do not penetrate into the cell. Virus penetration was detected by EM in control cells 3-6 hr after infection, and EB nuclear antigen was detectable in these cells 7-10 hr after infection. These results indicate that a special control mechanism at the virus-penetration level may play an important role in EBV-cell interactions and that the presence of EBV receptors on the cell surface may only be a preliminary condition for the infection of a cell by EBV. (15 refs.)

- 77-2052 **Epstein-Barr Virus DNA Synthesized in Superinfected Raji Cells.** (Eng.) Shaw, J. E. (Cancer Res. Center, Sch. Medicine, Univ. North Carolina, Chapel Hill, NC 27514) Seebeck, T.; Li, J. L.; Pagano, J. S. *Virology* 77(2): 762-771; 1977.

The DNA synthesized in Raji cells that were superinfected with virus from P3HR-1 cells [both of which are established Burkitt's lymphoma-derived cell lines that carry the Epstein-Barr virus (EBV) genome] was isolated and characterized. Radiochemically pure  $^{32}\text{P}$ -labeled viral DNA was obtained directly from the infected cells following a single purification on cesium chloride. Raji cells infected with P3HR-1 virus and labeled with  $^{32}\text{P}$  10 hr after infection synthesized only viral DNA; little if any viral DNA was synthesized by mock-infected Raji cells. About 90% of the viral DNA was localized in the nucleus of the superinfected cells after a 10-hr labeling period. The  $^{32}\text{P}$ -labeled DNA from superinfected cells reassociated with the DNA from Raji, P3HR-1, and virus DNA, but not with the DNA from a lymphoblastoid cell line lacking the EBV genome. All of the fragments produced by digestion of EBV-DNA with the restriction endonuclease EcoRI were present in a similar digest of DNA from the superinfected Raji cells. The viral DNA synthesized during superinfection apparently represents the entire EBV genome, but it is not known whether the viral molecules synthesized after superinfection are copies of the input DNA and/or of the resident EBV genomes. (26 refs.)

- 77-2053 **Clinical and Virological Findings in Patients with Cytologically Diagnosed Gynecologic Herpes Simplex Infections.** (Eng.) Vesterinen, E. (Lab. Pathology and Cytology, I-II Depts. Obstetrics and Gynecology, Univ. Central Hosp. Helsinki, Helsinki, Finland) Purola, E.; Saksela, E.; Leinikki, P. *Acta Cytol (Baltimore)* 21(2): 199-205; 1977.

Women with cytologically diagnosed herpes simplex virus (HSV) infections were analyzed in detail. Of 57,117 Papanicolaou (Pap) smears collected, 90 from 85 patients showed typical alterations of HSV infection, an overall frequency of 0.16%. The predominant morphological alteration

was the presence of "ground-glass" nuclei. This condition was characterized by nuclear homogenization, chromatin margination, and sometimes empty nuclei with an accentuated nuclear membrane. These changes occurred in isolated mononucleated epithelial cells or in multinucleated giant cells and were seen mostly in the ectocervical part of the smears. Positive virus isolation was obtainable only within a limited period from the positive Pap smear and was most successful when performed simultaneously with or within a week of cytologic diagnosis. Serologic samples were collected from 29 patients; no complement-fixing (CF) HSV antibodies were absent if the serum sample was taken early in the course of the clinically manifest disease. Gonorrhea was discovered in 13% of the HSV group but in only 5% of the control group. These results are in agreement with the venereal mode of transmission of herpes genitalis. (18 refs.)

- 77-2054 **Regulation of Herpesvirus Macromolecular Synthesis. VI. Synthesis and Modification of Viral Polypeptides in Enucleated Cells.** (Eng.) Fenwick, M. (Marjorie B. Kovler Viral Oncology Labs., Univ. Chicago, Chicago, IL 60637) Roizman, B. *J Virol* 22(3): 720-725; 1977.

Viral protein synthesis was studied in intact Vero cells and in cells enucleated with cytochalasin B (10  $\mu\text{g}/\text{ml}$ ) after infection with herpes simplex virus 1 (30-50 plaque-forming units/cell). Infected intact and enucleated cells were labeled with  $^{14}\text{C}$ -amino acids, and their polypeptides were subjected to autoradiography after electrophoresis in polyacrylamide gels. When protein synthesis was blocked by cycloheximide (50  $\mu\text{g}/\text{ml}$ ) from the time of infection, messenger RNA for viral  $\alpha\text{S}$ -infected cell polypeptides (ICP) 4, 0, and 27 accumulated in the cytoplasm and was expressed after removal of both the drum and nucleus. A host protein, ICP 22, whose synthesis was stimulated in intact cells, was not made in the enucleated cells. Removal of the nucleus inhibited the normal modification of viral protein ICP 4 to a form that migrates more slowly in polyacrylamide gels. After enucleation at 2 hr postinfection, a number of viral  $\beta$  and  $\gamma$  proteins continued to be synthesized, starting at 20%-25% of the normal rates and declining with a half-time of about 2 hr. The synthesis of ICP 4 declined more rapidly, suggesting that it is switched off in the cytoplasm. (11 refs.)

- 77-2055 **Requirement of Host Cell RNA Polymerase II in the Replication of Herpes Simplex Virus in  $\alpha$ -Amanitin-Sensitive and -Resistant Cell Lines.** (Eng.) Ben-Zeev, A. (Lab. Molecular Virology, Hebrew Univ. -Hadassah Medical Sch., Jerusalem, Israel) Becker, Y. *Virology* 76(1): 246-253; 1977.

The replication of herpes simplex virus (HSV) in  $\alpha$ -amanitin ( $\alpha$ -am)-sensitive (BSC-1 and BHK-21) and  $\alpha$ -am resistant ( $\alpha$ -amr, BHK-T6-G<sub>1</sub>) cell lines was studied in the presence



and absence of  $\alpha$ -am. Viral DNA synthesis but not cellular DNA synthesis was prevented in BSC-1 cells treated with 1  $\mu$ g/ml  $\alpha$ -am and in BHK-21 cells treated with 5  $\mu$ g/ml  $\alpha$ -am. This indicates that the expression of viral functions was arrested in the  $\alpha$ -am-treated cells in vivo. In the  $\alpha$ -amr BHK-T6-G<sub>1</sub> cell line, replication of HSV was unaffected in the presence of  $\alpha$ -am and amphotericin B. Since  $\alpha$ -am was able to penetrate into BSC-1 and BHK-21 cells in the presence of amphotericin B, it is assumed that it also penetrated into the BHK-T6-G<sub>1</sub> cells that have an  $\alpha$ -am-resistant RNA polymerase II. At 1-10  $\mu$ g/ml,  $\alpha$ -am had no effect on the ability of the mutant cell line to support HSV replication. This eliminates the possibility that the virus codes for an  $\alpha$ -am-sensitive RNA polymerase for transcription of the viral genome in infected cells. Transcription of HSV DNA is therefore carried out by the cellular RNA polymerase II both in the  $\alpha$ -am-sensitive and -resistant cell lines. The possibility also exists, however, that the virus codes for subunits of the RNA polymerase or for protein factors that may be involved in regulating the host cell RNA polymerase II to recognize and transcribe HSV DNA. (14 refs.)

- 77-2056 Comparison of DNA Polymerase Activities Induced by Herpes Simplex Virus Types 1 and 2.** (Eng.) Purifoy, D. J. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77030) *Intervirology* 6(6): 356-366; 1975/76.

DNA polymerase activities induced in HEP-2 cells by herpes simplex virus type 1 (HSV-1) and HSV-2 were compared. The induction kinetics of salt-stimulated DNA polymerase activity was similar after infection with HSV-1 and HSV-2 at multiplicities of infection of 2-20 plaque-forming units/cell. De novo synthesis of protein and messenger RNA was required for DNA polymerase induction by both types of virus. The DNA polymerase activity of three strains of each type of virus was compared by its response to various salt concentrations and by heat inactivation. Strain-dependent but not type-dependent differences were found by both tests. Extensive cross-neutralization of polymerase activity was obtained with specific antisera. Three of the four antisera tested showed only slightly better neutralization of DNA polymerase activity induced by the homologous type virus. It is concluded that the DNA polymerase activities induced by HSV-1 and HSV-2 cannot be readily differentiated on the basis of thermostability, response to salt, or neutralization by antisera. (19 refs.)

- 77-2057 DNA-binding Proteins of Cells Infected by Herpes Simplex Virus Type 1 and Type 2.** (Eng.) Powell, K. L. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Texas Medical Center, Houston, TX 77030) Purifoy, D. J. *Intervirology* 7(4/5): 225-239; 1976.

Proteins showing affinity for DNA in herpes simplex virus type 1 (HSV-1) and HSV-2-infected HEP-2 cells were compared by DNA-cellulose chromatography and polyacrylamide gel electrophoresis (PAGE). Virus-infected cells were labeled with a <sup>14</sup>C-amino acid mixture, and DNA-binding proteins were extracted by high-salt extraction. HSV-1 and HSV-2 inhibited the production of most of the host-cell DNA-binding proteins in infected cells. These proteins were replaced by a much simpler group of DNA-binding proteins that must be virus-specified or must represent a special group of host proteins selectively synthesized after virus infection. Almost no polypeptides of the same apparent molecular wt were found in HSV-2- and HSV-1-infected cells. Several examples of analogous polypeptides, however, were present in cells infected by both virus types. Proteins showing the highest affinity for DNA-cellulose were similar in molecular size in cells infected by both virus types. (16 refs.)

- 77-2058 Transcription of the Genome of Adenovirus Type 12. IV. Maps of Stable Late RNA from Productively Infected Human Cells.** (Eng.) Scheidtmann, K. H. (Inst. Genetics, Univ. Cologne, Cologne, W. Germany) Doerfler, W. *J Virol* 22(3): 585-590; 1977.

Human KB cells growing in suspension culture were infected with adenovirus type 12 (Ad12) at multiplicities of 100 plaque-forming units/cell. Virus-specific messenger RNA (mRNA) and stable nuclear RNA were isolated 42 hr after infection and mapped on the viral genome through the use of restriction endonuclease fragments of Ad12 DNA. Late after infection, the r (rightward) DNA strand was transcribed into stable nuclear RNA, but the l (leftward) strand was expressed only to a minor extent. Ad12-specific mRNA originated from the following sections on the viral genome: 0 to 0.11, 0.18 to 0.20, 0.27 to 0.49, 0.56 to 0.63, 0.68 to 0.84, and 0.89 to 0.92 fractional length units on the r strand and 0.11 to 0.16, 0.22 to 0.27, 0.50 to 0.54, 0.62 to 0.66, 0.855 to 0.865, and 0.93 to 1.0 fractional length units on the l strand. Self-complementary viral RNA isolated at 8 hr postinfection was complementary to about 20% of each strand of the viral genome, and that isolated at 42 hr annealed to 70%-80% of each DNA strand. (16 refs.)

- 77-2059 Studies of the UV-Sensitivity of Virus-Specific RNA Synthesis in Cells Infected or Transformed by Adenovirus 5 and SV40.** (Eng.) Mantieva, V. L. (Inst. Molecular Biology, Acad. Sciences of the USSR Moscow, B-312, USSR) Chumakov, P. M.; Koslov, Y. V.; Shelov, A. A.; Zalmanzon, E. S.; Savina, A. A. *Mol Biol Rep* 3(3): 243-249; 1977.

UV radiation-induced inhibition of host and virus-specific RNA synthesis was studied and compared in human and Adenovirus 5 (Ad5)-infected KB cells. Ad5-infected cells

were irradiated with UV light (254 nanometers) and incubated with nonirradiated control cells in a mixture of radioactively-labelled RNA precursors. After various incubation periods, the RNA was extracted from the cells, purified, and analyzed via sedimentation on sucrose gradients and hybridized to DNA fragments by nitrocellulose filter binding assays. It was shown that the total content of virus-specific RNA in the newly-formed and labelled RNA is not significantly changed as compared with intact cells and that virus-specific RNA synthesis is more resistant to UV radiation than the host RNA synthesis. The data support the idea that UV irradiation produces stop points but does not interfere with the initiation process in the transcription of DNA. Transcription of the B fragment is inhibited to a much greater extent by UV irradiation than is the A fragment and the size of the host transcriptions is close to that of the adenovirus genome. (21 refs.)

77-2060    **Localization of the SV40 T Antigen in Hamster Cells Transformed by PARA(3ct)-Adenovirus 7.** (Eng.) Dottorini, S. (Inst. Infectious Diseases, Perugia Univ., Policlinico Montelucente, I-06100 Perugia, Italy) Tassi, C. *Intervirology* 6(6): 343-349; 1975/76.

The localization of the SV40 T antigen in the nuclear and/or cytoplasmic fraction of hamster cells transformed by the hybrid virus, PARA(3ct)-adenovirus 7, and by simian virus 40 (SV40) was examined by immunofluorescence and complement-fixation tests. The hamster H50 cell strain (transformed by SV40) and the P7-/BL/SV/3D strain [transformed by PARA(3ct)-adenovirus 7] showed a preferential localization of the SV40 T antigen in the nucleus. Two other PARA(3ct)-adenovirus 7-transformed hamster cell lines (P7-/Ar/d/15A and P7-/Ar/m/14A) showed an almost equal concentration of SV40 T antigen in the nucleus and cytoplasm. The H50, P7-/BL/SV/3D, and P7-/AR/m/14A cells were oncogenic for hamsters; the P7-/Ar/d/15A line was not. It is not known why the same virus transforms hamster cells in two different and stable phenotypes. It is hypothesized, however, that the blockage of transport into the nucleus of SV40 T antigen, associated with the PARA(3ct)-adenovirus genome, must be partially or completely overcome as a prerequisite for transformation. This may explain the low oncogenicity of PARA(3ct) adenovirus 7 and point to a role of SV40 T antigen in transformation through its action on nuclear functions. (18 refs.)

77-2061    **Distinct Nonstructural Polypeptides in Polyoma and Simian Virus 40 DNA-Protein Complexes.** (Eng.) Qureshi, A. A. (Departement Microbiologie, Faculte Medecine, Universite Sherbrooke, Sherbrooke, Quebec, Canada J1H 5N4) Bourgaux, P. *Virology* 77(1): 418-420; 1977.

It has been recently reported that the 55S-sedimenting viral complex extracted with Triton V-100 from polyoma (Py)-infected cells contains, in addition to the virus-coded structural polypeptides, a polypeptide absent from the virion, with an apparent molecular wt (MW) of  $\geq 70,000$ . An analogous nonstructural polypeptide in a viral complex similarly isolated from simian virus 40 (SV40) is now reported. The viral DNA-protein complex was extracted from CV<sub>1</sub> cells infected with SV40. Migration in sodium dodecyl sulfate-polyacrylamide gels indicated MW of 76,000 and 86,000, respectively, for the SV40 and the Py virus polypeptide. These observations may help to define a practical approach for the purification and characterization of some of the proteins, either cellular or viral, involved in virus replication. (28 refs.)

77-2062    **Uncontrolled Nuclear Division in Murine Cells Abortively Transformed by Simian Virus 40.** (Eng.) O'Neill, F. J. (Dept. Res., Veterans Admin. Hosp., Salt Lake City, UT 84113) *Intervirology* 7(3): 145-154; 1976.

Mouse cells abortively transformed by simian virus 40 (SV40) were examined for uncontrolled nuclear division using cytochalasin B (CB), which prevents cytoplasmic cleavage without directly affecting nuclear division. 3T3 cells and mouse embryo fibroblasts (MEF) were treated with SV40 at inputs of 500 or 25 plaque-forming units/cell for 90 min and then incubated in medium containing 1.5  $\mu\text{g/ml}$  CB for 7 days. Controls received SV40 alone or CB alone. MEF or 3T3 cells showed controlled nuclear division when treated with CB. CB-treated transformed cells, however, showed uncontrolled nuclear division, and they became highly multinucleated. These highly multinucleated cells may represent abortively transformed cells, since the actual number of focus-forming units of SV40 was too low to account for the appearance of these cells. When CB treatment was begun 6 days after SV40 inoculation, the large increase in highly multinucleated cells was not observed. It is not clear why some stably SV40-transformed 3T3 cells maintain controlled nuclear division while abortively transformed 3T3 cells show uncontrolled nuclear division. Perhaps many stably transformed 3T3 cells do not have all their possible integration sites occupied and, therefore, have a relatively small number of viral genomes per cell. (20 refs.)

77-2063    **Low Molecular Weight Nuclear RNA from SV40-transformed WI38 Cells; Effect on Transcription of WI38 Chromatin In Vitro.** (Eng.) Krause, M. O. (Dept. Biology, Univ. New Brunswick, Fredericton, N. B., Canada E3B 5A3) Ringuette, M. *Biochem Biophys Res Commun* 76(3): 796-803; 1977.

Nuclei isolated from normal and simian virus 40 (SV40)-



transformed WI38 cells were used as templates for RNA synthesis *in vitro*. Comparison of template properties showed marked differences between the two chromatins with both homologous and heterologous enzymes. Addition of the 0.35 M NaCl extract from the chromatin of the SV40-transformed cells revealed two separate activities: one stimulating the template activity of normal WI38 chromatin and one affecting the stability of the homologous RNA polymerase. Separation of the protein and low-molecular-wt nuclear RNA (SnRNA) fractions in the extract demonstrated that the stabilization effect is due to chromosomal protein(s). However, SnRNA alone was found to be responsible for stimulation of the transcriptional activity of normal cells to a level indistinguishable from that of the transformed cells. (27 refs.)

- 77-2064 **Simian Virus 40 Gene A Regulates the Association Between a Highly Phosphorylated Protein and Chromatin and Ribosomes in Simian Virus 40-transformed Cells.** (Eng.) Segawa, K. (Dept. Tumor Virus Res., Inst. Medical Science, Tokyo 108, Japan) Yamaguchi, N.; Oda, K. *J Virol* 22(3): 679-693; 1977.

The species of proteins associated with chromatin and ribosomes of simian virus 40 (SV40)-transformed and untransformed C3H/He mouse kidney cells, Fisher rat embryo fibroblast cells, and African green monkey kidney cells were compared by sodium dodecyl sulfate-polyacrylamide gel electrophoresis after *in vitro* or *in vivo* phosphorylation of the proteins. *In vitro* phosphorylation was carried out by the protein kinase associated with these organelles and  $\gamma$ - $^{32}$ P-ATP as the phosphoryl donor. The reaction products contained both phosphoserine and phosphothreonine in approx equal amounts. Electrophoresis indicated that a highly phosphorylated protein with a molecular wt of 90,000 daltons (90K protein) is associated with the chromatin and ribosomes from transformed cells but not from untransformed cells. The 90K protein could be extracted with 0.5-1.0 M NaCl or KCl; it remained associated with the runoff ribosomes prepared by the puromycin reaction of the postmitochondrial supernatant in the protein-synthesizing system. *In vitro* phosphorylation of chromatin and ribosomes from mouse and rat cells transformed by a temperature-sensitive (tsA) SV40 mutant indicated a marked decrease in 90K protein in cells cultivated at the restrictive temperature. A similar temperature-dependent decrease in phosphorylated 90K protein occurred in nonhistone chromosomal and ribosome-associated with protein fractions from SV40 tsA-transformed cells labeled with  $^3$ H-leucine and  $^{32}$ P-orthophosphate *in vivo*. These results indicate that the SV40 A gene is closely related to the synthesis and/or binding of the 90K protein. Although the molecular wt of this protein is similar to that of SV40 T antigen, it was not immunoprecipitated with anti-SV40 T sera. (45 refs.)

- 77-2065 **Electrophoretic Properties of Temperature-sensitive Mutant Particles of Simian Virus 40.**

(Eng.) Zweig, M. (Frederick Cancer Res. Center, Post Office Box B, Frederick, MD 21701) *Intervirology* 6(6): 350-355; 1975/76.

Forty-five temperature-sensitive strains of simian virus 40 (SV40) particles belonging to four complementation groups of the late region were examined by agarose gel electrophoresis to identify the SV40 gene(s) coding for the surface components of the virion. Five strains (B204, B218, B228, B233, and BC226), which are members of the B and C complementation groups, yielded virions with aberrant mobilities. Previous genetic mapping had suggested that B and BC mutations are in a single gene. The virions of these five electrophoretic mutants had greater positive charges than wild-type particles. The apparent absence of temperature-sensitive mutations leading to virions with a greater negative charge may be due to the possibility that such mutations are lethal rather than conditionally lethal. These aberrant electrophoretic properties can be accounted for by mutations causing substitutions by more positively charged amino acid residues at the virion surface. The findings suggest that the product of genes B and BC is a significant component of the SV40 capsid. (5 refs.)

- 77-2066 **Characterization of Simian Virus 40 tsA58 Transcriptional Intermediates at Restrictive Temperatures: Relationship Between DNA Replication and Transcription.** (Eng.) Birkenmeier, E. H. (Institut de Recherches Scientifiques sur le Cancer, Villejuif, France) May, E.; Salzman, N. P. *J Virol* 22(3): 702-710; 1977.

Levels of viral transcriptional activity in African green monkey kidney cells infected with a temperature-sensitive mutant (tsA58) of simian virus 40 (SV40) were determined after Sarkosyl extraction. This procedure yields transcriptional complexes that contain RNA polymerase H and a small number of other proteins. When cells infected with tsA58 virions at 33 C were shifted to the nonpermissive temperature of 40 C between 18-48 hr postinfection, DNA synthesis was completely inhibited within 45 min. The RNA polymerase activity continued to increase for 5 hr before reaching a plateau. The RNA synthesized from both early and late SV40 DNA strands increased after the shift, and there was a threefold increase in the ratio of early-to-late RNA species. No comparable increase in polymerase activity or RNA synthesis occurred when cells infected with wild-type SV40 were shifted from 33 to 40 C; at 33 C, the relative amount of RNA transcribed from the wild-type early DNA strand was less than tsA58 at 33 C. The tsA58 transcriptional complexes extracted from cells grown at 33 C sedimented heterogeneously in sucrose gradients, with a peak near 26S. The results indicate that continued synthesis of viral DNA is not a prerequisite for maintenance of late viral transcription and that sedimentation of the transcriptional complex at 26S is not related to actively replicating DNA molecules serving as templates for transcription. The increase in the synthesis of early and late RNA at the restrictive temperature without concurrent DNA synthesis is discussed in relationship to the function of the A gene product. (20 refs.)

7-2067 **Structure and Biology of Minicircular Simian Virus 40 DNA (Meeting Abstract).** (Eng.) Chowdhury, K. (Deutsches Krebsforschungszentrum, Inst. fur Virusforschung, Im Neuenheimer Feld 280, D-6900 Heidelberg 1, W. Germany) Ruben-Barretto, M.; Sauer, G. *Hoppe Seylers Z Physiol Chem* 358(3): 224; 1977. (no refs.)

7-2068 **Mutagenesis by an Animal Virus. Characterization of Some Azaguanine Resistant Chinese Hamster Cell Mutants Selected After SV40 Infection (Meeting Abstract).** (Eng.) Theile, M. (Zentralinstitut fur Molekularbiologie der AdW der DDR, Abt. Zellgenetik,

Berlin-Buch, E. Germany) Strauss, M. *Mutat Res* 46(3): 239; 1977. (2 refs.)

\* (Review): 77-1820, 77-1821, 77-1822, 77-1823, 77-1824, 77-1825, 77-1826, 77-1827, 77-1828, 77-1883.

\* (Phys): 77-1970, 77-1971, 77-1972, 77-2001.

\* (Immun): 77-2069, 77-2079, 77-2083, 77-2084, 77-2087, 77-2089, 77-2093, 77-2102, 77-2105, 77-2110, 77-2111, 77-2114, 77-2115, 77-2116, 77-2117, 77-2120, 77-2121, 77-2123, 77-2136, 77-2142, 77-2169, 77-2191, 77-2193, 77-2195, 77-2197, 77-2200, 77-2201, 77-2202, 77-2203, 77-2204, 77-2205, 77-2206, 77-2207, 77-2224, 77-2228.

\* (Path): 77-2239, 77-2249, 77-2250, 77-2256.

\* (Epid): 77-2290.



- 77-2069 "Ganglioprotein and Globoprotein": The Glycoproteins Reacting with Anti-ganglioside and Anti-globoside Antibodies and the Ganglioprotein Change Associated with Transformation. (Eng.) Tonegawa, Y. (Biochemical Oncology, Fred Hutchinson Cancer Res. Center, 1124 Columbia, Seattle, WA 98104) Hakomori, S. *Biochem Biophys Res Commun* 76(1): 9-17; 1977.

The cell-surface-labeled glycoproteins that are reactive to antiglycolipid antibodies were solubilized by zwitterionic or nonionic detergents and separated by specific antiglycolipid antibodies through double immuno precipitation or specific absorption on *Staphylococcus aureus*. The patterns of these glycoproteins were examined by sodium dodecyl sulfate (SDS)-acrylamide gel electrophoresis and compared to those of glycolipids. The pattern of the surface-labeled globoside in human RBC consisted of four fractions, the major one being coincident with the glycolipid band. The other three peaks were globoproteins 2, 3, and 4. Globoprotein 3, the major globoprotein of the RBC, was in the PAS 2 or 3 region. The GM<sub>1</sub> ganglioproteins of 3T3 cells were all deleted in Kirsten murine sarcoma virus transformants. The intense protein peaks reactive to antisialo GM<sub>2</sub> (ganglio-N-triosylceramide) appeared in these transformants. The results indicate the presence of cell-surface glycoproteins that carry the same carbohydrate chain as those found in the major glycolipids such as globoside and GM<sub>1</sub> ganglioside. (32 refs.)

- 77-2070 Reversible Blocking of Antibody-dependent Cell-mediated Cytotoxicity. (Eng.) Sugamura, K. (Immunobiology Res. Center, Univ. Wisconsin, Madison, WI 53706) Smith, J. B. *Cell Immunol* 30(2): 353-357; 1977.

The ability of human peripheral blood lymphocytes to kill antibody-coated Chang liver cells in antibody-dependent cell-mediated cytotoxicity (ADCC) was studied. Specific blocking of ADCC with aggregated immunoglobulin G (agg-IgG) was determined using effector cells pretreated with 0-20 mg/ml of agg-IgG, aggregated human serum albumin (agg-HSA), or aggregated bovine serum albumin (agg-BSA). Chang liver cells labeled with <sup>51</sup>Cr were used as target cells. Effector cells ( $1 \times 10^6$ ) were then mixed with target cells ( $1 \times 10^4$ ) and incubated. Cytotoxicity was determined by release of radioactivity from the target cells. Treatment of the cells with 20 mg/ml of agg-IgG reduced killing in ADCC by 60%-79%, and the response was dose-dependent. Agg-HSA and agg-BSA did not affect killing in ADCC. If ADCC occurs in vivo, as it did in these studies, it may be possible to restore blocked effector-killer cell function by reducing the concentration of immune complexes in the circulation. (23 refs.)

- 77-2071 Genetic Control of Antibody Variable Regions. (Eng.) Weigert, M. (Inst. Cancer Res., Philadelphia, PA 19111) Riblet, R. *Cold Spring Harbor Symp Quant Biol* 41: 837-846; 1977.

The remarkably simple variability patterns for  $\lambda$  chains produced by inbred mouse myeloma tumors indicate that there is a single germ-line gene coding for each complete-variable N-region subgroup that is generated somatically. The locations of variability in  $\lambda$  amino acid sequences suggest that the variants are selected sequentially by antigen. Max likelihood estimates of the total number of V<sub>K</sub> subgroups (genes) of NZB and BALB/c mice are given. Identical light (VL) or heavy (HV) regions from independently induced tumors serve to identify the product of a germ-line gene. If the products of VL and VH genes interact in all combinations, the number of germ-line antibodies would be in the range of  $10^4$ - $10^5$ . Several examples of naturally occurring germ-line antibodies can be identified tentatively. Detailed analysis of the VHDEX gene has revealed interesting anomalies. These may reflect the influence of multiple, homologous loci, such as those coding for VL or VH subgroups, on the recombination rate and might suggest mechanisms by which V-region translocation occurs. (36 refs.)

- 77-2072 Suppression of Myeloma Establishment in Adult Mice with Anti- $\mu$  Antiserum. (Eng.) Douglas, J. G. (Dept. Medical Microbiology, Univ. Wisconsin, Madison, WI 53706) Manning, D. D. *Cell Immunol* 30(1): 100-107; 1977.

The effect of anti- $\mu$  antibodies on the in vivo development of transplanted MOPC-104E tumors in adult BALB/c and nude mice was examined by direct neutralization and passive immunization. In the first approach, in vitro preincubation of  $2.3$  to  $3.0 \times 10^5$  viable MOPC-104E cells in 0.1 ml phosphate-buffered saline with 0.1 ml anti- $\mu$  antiserum and subsequent injection of the cell-antiserum mixture into mice suppressed tumor development in 13/24 mice. In the second approach, mice that received a single 0.4-ml ip injection of anti- $\mu$  1 day before tumor implantation uniformly failed to develop tumors (0/22). Anti- $\mu$  antiserum exhibited virtually no cytotoxicity against MOPC-104E cells in vitro. Antiserum prepared against the whole plasmacytoma cell, although highly cytotoxic in vitro, was not as effective a myeloma inhibitor as was anti- $\mu$  antiserum. These results suggest that neither complement-mediated cytotoxicity nor T-cell-mediated immunity is the primary mechanism for anti- $\mu$  mediated suppression of myeloma development. The most likely

explanations are macrophage arming, antibody-dependent cell-mediated cytotoxicity, and/or opsonization. (17 refs.)

77-2073    **Serum Immunoglobulins and C<sub>3</sub> Complement Concentrations in Malignancy.** (Eng.) Edwards, A. J. (Surgical Unit, Whipps Cross Hosp., London, England) Lee, M.; Harcourt, G. *Clin Oncol* 3(1): 65-73; 1977.

A series of common immunoglobulins (IgA, IgG, IgM, and IgE) and C<sub>3</sub> complement were assayed in patients with benign and malignant gastrointestinal (GI) lesions. Of the 36 patients who participated, 24 had nonmetastasized carcinoma and 11 had nonmalignant conditions (controls). IgA, IgG, IgM, and C<sub>3</sub> were measured in systemic, portal, and local sera by radial immunodiffusion, and IgE was assayed by solid-phase radioimmunoassay. No differences were seen in Ig concentration at any of the locations, nor was there any significant difference between corresponding Ig levels in the malignant benign conditions. However, the ratio of IgA to IgG was raised ( $p < 0.05$ ) in all three sera in all cancer patients (systemic 0.26, portal 0.26, local 0.26), compared to control values of 0.19, 0.19, and 0.18. C<sub>3</sub> levels (mg/100 ml) were also elevated ( $p < 0.01$ ) in all cancer patients (systemic 140.3, portal 145.3, local 148.1), compared to 107.0, 112.6, and 112.7 for controls. These findings show no preferential absorption or secretion of the local production of these Ig's by the tumor. The elevated C<sub>3</sub> levels suggest a locally produced tumor effect. (50 refs.)

77-2074    **Double Immunoglobulin Production in Cloned Somatic Cell Hybrids Between Two Human Lymphoid Cell Lines.** (Eng.) Rosen, A. (Dept. Tumor Biology, Karolinska Institutet, Fack, S-104 01 Stockholm, Sweden) Clements, G.; Klein, G.; Zeuthen, J. *Cell* 11(1): 139-147; 1977.

Somatic cell hybrids between two human lymphoid cell lines, Raji and Namalwa, were examined for surface immunoglobulin expression. The majority of hybrids expressed both parental phenotypes. The expression of kappa light chains did not exclude the expression of lambda light chains in the hybrids. These results provide evidence that the mutual exclusion of kappa and lambda in immunoglobulin producing cells does not proceed through the continuous production of diffusible repressing factors. (28 refs.)

77-2075    **Position of a Light Chain Tyrosyl Residue in the Combining Site of a Rabbit Anti-p-azobenzoate Antibody.** (Eng.) Lee, Y. (Dept. Immunology Res., Roswell Park Memorial Inst., New York State Dept. Health, 666 Elm St., Buffalo, NY 14263) Roholt, O. A.; Appella, E.; Pressnan, D. *Immunochemistry* 14(4): 269-276; 1977.

The isolation and characterization of chymotryptic peptides containing a tyrosyl residue from the combining site of a rabbit anti-p-azobenzoate antibody preparation of restricted

heterogeneity were achieved by paired iodination. The high-ratio peptides C-1-H, C-2-L, and C-3-L were obtained. Peptide C-3-L, which is composed of 11 amino acid residues, came from the light chain of the antibody. Upon amino acid analysis, peptide C-2-L was noted to yield threonine, two serines, and one glutamic acid, valine, and tyrosine. Peptide C-1-H was derived from the heavy chain of the pair-iodinated antibody, and amino acid analysis gave aspartic acid, serine, tyrosine, and three glycines. The sequence of the first 38 residues from the N-terminus of the light chain was determined, and the combining-site tyrosyl residue was demonstrated to be at position 30 of the light chain, in the first hypervariable region. The latter fact was demonstrated by the finding that 14% of the applied <sup>125</sup>I radioactivity in the radioiodinated light chain was recovered at the 30th cycle of degradation. An antibody site reacting with a particular structure may be specified by different sequences and the common presence of a given residue in a particular hypervariable position. (38 refs.)

77-2076    **The Primary Structure of a Human Immunoglobulin L-Chain (K-Type, Subgroup II): Bence Jones Protein NIM.** (Eng.) Eulitz, M. (Institut für Hamatologie, Abteilung Immunologie der Gesellschaft für Strahlen-und Umweltforschung mbH, Landwehrstr. 61, 8000 München 2, W. Germany) Kley, H. P. *Immunochemistry* 14(4): 289-297; 1977.

The nearly complete amino acid sequence of Bence Jones protein Nim (from a patient suffering from plasmacytoma) was reconstructed from a study of the tryptic and chymotryptic peptides of the aminoethylated or carboxymethylated polypeptide chain. By combining manual and automatic degradation, it was possible to construct the almost entire primary structure of the protein. The protein contained 219 amino acids, 114 of which belong to the variable part and 105 to the constant part. A constancy of two amino acids within the first hypervariable region into subgroup II of human  $\kappa$ -L-chains was found. In the variable part, five extra residues were observed, which, together with the subgroup-specific positions, were characteristic for subgroup  $\kappa$ -II. With the exception of the allotype-related position 191, where valine was seen, no amino acid exchange was noted in the constant part. Considering the results, it appears necessary to analyze homogeneous human immunoglobulins derived from other sources, such as from patients suffering from multiple myeloma. (45 refs.)

77-2077    **A Study of the Variable Heavy Chain (VH) Region of Membrane-bound Ig on Human Chronic Leukemic Lymphocytes.** (Eng.) Forre, O. (Inst. Immunology and Rheumatology, Rikshospitalet Univ. Hosp., Oslo, Norway) Froland, S. S.; Natvig, J. B.; Michaelsen, T. E.; Johnson, P. M.; Ly, B.; Laake, K. *J Immunol* 118(5): 1513-1516; 1977.

The membrane-bound immunoglobulin (Ig) of lymphocytes from 20 patients with chronic lymphocytic leukemia (CLL)



was analyzed by direct immunofluorescent staining using anti-F(ab')<sub>2</sub> and heavy-chain variable (VH) subgroup-specific antisera. Using anti-F(ab')<sub>2</sub>, high percentages (30%-100%) of lymphocytes with membrane-bound Ig were found in all leukemias investigated, indicating their probable B-cell origin. CLL cells from each patient stained for only one subgroup; the ratio of CLL lymphocytes staining with anti-VH1, anti-VH11, and anti-VH111 subgroup antisera was 6:7:7, respectively. This restriction represents further evidence for the monoclonality of leukemic cell proliferation. Indirect immunofluorescent studies were also performed on some of the lymphocytes using an antiserum raised against IgG, myeloma protein (Kup). Cells staining with anti-F(ab')<sub>2</sub> antiserum also stained with anti-VH antiserum, and the percentages of lymphocytes stained with each were closely similar. Thus, the same cells, ie, B lymphocytes, were stained with both antisera. (20 refs.)

- 77-2078 Murine Plasma Cells Secreting More Than One Class of Immunoglobulin Heavy Chain. II. SAMM 368--A Plasmacytoma Secreting IgG2b- $\kappa$  Immunoglobulins Which Do Not Share Idiotype Determinants.** (Eng.) Morse, H. C. (Building 5, Room 224, NIH, Bethesda, MD 20014) Neiders, M. E.; Lieberman, R.; Lawton, A. R.; Asofsky, R. *J Immunol* 118(5): 1682-1689; 1977.

Immunofluorescence studies of ascites cells from SAMM 368, a BALB/c plasmacytoma secreting IgA- $\kappa$  and IgGwb- $\kappa$  paraproteins, were made. The tumor was established and maintained in transplantation by injection of ascites cells into BALB/c mice conditioned with a single ip injection of pristane given 7-30 days earlier. Purified, heavy-chain, class-specific antiglobulins were used in the studies. The results showed that single cells of the plasmacytoma contained immunoglobulins (Ig) of both the IgA and IgG2 classes (heavy chains). Although heterologous antisera identified the paraprotein as IgG2b, SAMM 368 IgG2b carried no detectable allotypic determinants. IgA bore the BALB/c A determinants, as demonstrated by analysis of the purified paraproteins for allotype markers. The two paraproteins produced by the tumor did not share idiotype determinants, as indicated by idiotype antisera prepared in rabbits and mice. Further evaluation of the plasmacytoma and its products may be instructive for understanding regulation of Ig genes, clonal expansion, and the chemical nature of allotype markers. (59 refs.)

- 77-2079 Induction of Immunoglobulin Synthesis in Abelson Murine Leukemia Virus-transformed Mouse Lymphoma Cells in Culture.** (Eng.) Weimann, B. J. (Basel Inst. Immunology, CH 4058 Basel, Switzerland) *Cold Spring Harbor Symp Quant Biol* 41: 163-164; 1977.

When Abelson murine leukemia virus (A-MuLV)-transformed tissue culture cells derived from BALB/c mice were grown in the presence of 1% dimethyl sulfoxide (DMSO) for 36 or 60 hr, cell differentiation and immuno-

globulin (Ig) synthesis could be induced. Approximately 90% of the cells of cell line K grown in the presence of DMSO for 60 hr carried Ig on their membranes, and 100% expressed Fc receptors. In addition to the immunofluorescence procedures employed, Ig synthesis could be observed by biosynthetic incorporation of radioactive leucine into Ig characterized by immunoprecipitation. The results indicate that cells are stimulated to produce IgM molecules and large amounts of an unidentified protein that binds to immune complexes. (17 refs.)

- 77-2080 Immune Responses of BALB/c Mice to the Idiotype of T15 and of Other Myeloma Proteins of BALB/c Origin: Implications for an Immune Network and Antibody Multispecificity.** (Eng.) Sakato, N. (Res. Inst. Microbial Diseases, Osaka Univ., Yamada-kami, Suita, 575, Osaka, Japan) Janeway, C. A.; Eisen, H. N. *Cold Spring Harbor Symp Quant* 41: 719-724; 1977.

Sterilized alloantiserum (CE/J anti-T15, 0.05 ml) injected ip 3 x/wk for 3 wk into four newborn BALB/c mice suppressed the production of immunoglobulin (Ig) with the natural T15 myeloma protein (IgA/k) idiotype. Three days after the final injection and at 1-wk intervals thereafter, mice received a total of six injections of 100  $\mu$ g T15. During this time newly produced anti-T15 appeared in all four mice. Antibodies to T15 were also formed in 8-wk-old germ-free BALB/c mice by injections of 100  $\mu$ g T15 at various intervals. The anti-T15 molecules were clearly anti-idiotypes in both cases, because substantial inhibition by phosphorylcholine could be demonstrated. Spleen cells from mice making antibodies to the T15 idiotype enhanced the production of anti-DNP (2,4-dinitrophenyl) antibodies in response to DNP<sub>4</sub>-T15. The possibility of a reciprocal relationship between the abundance and immunogenicity of Ig idiotypes in an immune network is discussed. (24 refs.)

- 77-2081 Marrow-dependent (M) Cell Control of Responses to Oncogenic and Non-oncogenic Infections.** (Eng.) Bennett, M.; Yonkosky, D.; Kumar, V.; Eastcott, J. W.; Chromey, N.; Baker, E. E. In: *Immuno-aspects of the Spleen. Proceedings of a Conference on Immunodynamics held in Cleveland, Ohio, May 19-20, 1976*. Battisto, J. R.; Streilein, J. W., eds. (New York: North-Holland Publishing Co.): pp. 345-356; 1976.

The role of marrow-dependent (M) cells in genetic resistance against Friend leukemia virus (FV) and resistance to early stages of infection by the bacterium *Listeria monocytogenes* (LM) is discussed. Resistance to the immunosuppressive and leukemogenic effects of FV were observed in adult C57BL/6 (B6), B10.D2n/Sn (B10.D2), and C58 mice. Treatment of B6 mice with <sup>89</sup>Sr destroyed their M cells, and they subsequently became susceptible to leukemia induction and immunosup-

pression by FV. Treatment of these mice with agents that destroy T- and B-cell functions failed to abrogate their resistance to FV. M-cell-deficient neonatal B6 and B10.D2 mice did not demonstrate resistance to the leukemogenic effects of FV. The effects of a variety of immunoregulatory agents on the growth of iv-injected LM during the first 2 days of infection were tested to analyze further the role of M cells and macrophages. Silica, BALB/c anti-c57BL spleen cell serum (ASCS), and cyclophosphamide (CP) nullified the resistance to allogeneic marrow stem cells, but azathioprine, cytosine arabinoside (ara-C), and cortisol did not. Further studies with (C57BL x DBA/2)F1 mice pretreated with immunoregulatory agents and exposed to lethal total-body irradiation showed that silica,  $^{90}\text{Sr}$ , antilymphocytic serum, and azathioprine enhanced their susceptibility to LM. Cortisol and ara-C had no effect, and cyclophosphamide, ASCS, and *Corynebacterium parvum* organisms increased their resistance to LM. The results suggest that M cells are different from macrophages. They may be a heterogeneous population, with all members requiring a marrow microenvironment for functional differentiation, but with differential sensitivity to various immunoregulatory agents. (21 refs.)

- 77-2082 **Regulatory Mechanisms in the Immune Response to Cell-Surface Antigens.** (Eng.) Lake, P. (Imperial Cancer Res. Fund Tumour Immunology Unit, Univ. Coll., London, W.C.1, England) Mitchison, N. A. *Cold Spring Harbor Symp Quant Biol* 41: 589-595; 1977.

The adoptive secondary response was used to analyze the associative recognition, or help, that is mediated by TH (thymus helper) cells in response to the major histocompatibility antigens of the mouse, H-2D and H-2K, and to the minor alloantigen Thy-1. A study using a naturally occurring A-strain recombination indicated that antigens of the major histocompatibility complex and, possibly, H-2D alone appear to provide the determinants toward which TH help is directed, thus demonstrating intramolecular help. In another experiment, B cells received help for the anti-H-2D response from minor alloantigens, thus demonstrating intermolecular help. H-2 inhibition of the anti-Thy-1 adoptive secondary response is apparently mediated by intermolecular, intrastuctural competition. This result supports the view that separate surface molecules are coprocessed by the immune system. Thy-1 can induce and direct an in vitro antibody response to an alloantigen. Responses in cancer cells may be manipulated in the future by associative help or inhibition. (21 refs.)

- 77-2083 **Immune Response of Athymic Nude Mice to Papovavirus SV40 Tumor-associated Antigens.** (Eng.) Tevethia, S. S. (Dept. Pathology and Cancer Res. Center, Tufts Univ. Sch. Medicine, Boston, MA 02111) Waneck, G.; Tevethia, M. J. *Int J Cancer* 19(5): 700-706; 1977.

The requirement of T-cell functions in the induction of an immune response to simian virus 40 (SV40)-specific tran-

splantation rejection antigen and intranuclear tumor antigen was studied in athymic nude mice. Athymic nude mice immunized with two sc injections of  $1 \times 10^7$  plaque-forming units of SV40 were unable to reject a challenge with either  $1 \times 10^5$  or  $1 \times 10^4$  tumor cells. Sensitized lymphocytes capable of inhibiting tumor growth in vivo could not be demonstrated in the spleens of virus-immunized mice. Athymic nude mice bearing tumors induced by virus-free SV40-transformed BALB/c cells failed to develop antibodies to intranuclear T antigen. Athymic nude mice also failed to respond to viral antigens. Thus, it is concluded that T-cell functions are required in the induction of a cellular immune response to SV40-specific transplantation rejection antigen and in the induction of a humoral immune response to SV40-specific T antigen and virion antigen. (22 refs.)

- 77-2084 **Cellular and Humoral Anti-tumor Immune Responsiveness in Chickens Bearing Tumors Induced by Avian Sarcoma Virus.** (Eng.) Wainberg, M. A. (Lady Davis Inst. Medical Res., Jewish General Hosp., Montreal, Quebec, Canada) Yu, M.; Schwartz-Luft, E.; Israel, E. *Int J Cancer* 19(5): 680-687; 1977.

A comparison was made of the abilities of normal chicken embryo fibroblast (CEF) cells, Rous sarcoma virus (RSV)-transformed CEF cells, avian Rous sarcoma (RS) tumor cells, and murine RS cells to serve as targets and antigen donors in various assays for the detection of cellular and humoral antitumor immunity in chickens bearing tumors induced by RSV. Cytotoxicity tests showed that avian and murine RS cells were more susceptible to the killing effects of sensitized lymphocytes than were transformed CEF, which in turn were more reactive than normal CEF. In contrast, sera from tumor-bearing animals were able to stain by indirect immunofluorescence only the avian RS and transformed CEF cells. Extracts of both transformed CEF cells and avian RS cells, but not normal CEF, were equally effective as inhibitors of the migration of peritoneal exudate cells derived from tumor-bearing animals. Transformed CEF produced far higher quantities of transforming virus progeny than avian RS cells, although the latter appeared able to synthesize defective viral particles. These data indicate that antitumor immunity in the avian sarcoma system can vary significantly, depending on the types of assays and target cells employed. (43 refs.)

- 77-2085 **Immune Status in Untreated Patients with Lymphoreticular Malignancy--a Multi-factorial Study.** (Eng.) Hancock, B. W. (Academic Div. Medicine, Royal Hosp., Sheffield, England) Bruce, L.; Sugden, P.; Ward, A. M.; Richmond, J. *Clin Oncol* 3(1): 57-63; 1977.

The cellular immune status of 102 untreated patients suffering from either localized or generalized malignant lymphomas is assessed. Techniques involving intradermal skin tests with various recall antigens, leukocyte migration, lym-



phocyte transformation, spontaneous E-rosette formation, and automated precipitation and determination of serum immunoglobulins, and neutrophil function determination were used to assess the cellular immune characteristics in patients with lymphomas of varying stages and involvement. Lower lymphocyte and higher neutrophil counts were observed in patients with widespread Hodgkin's disease or reticulum cell lymphoma. Humoral immunity, as assayed via automated immunoprecipitation of serum immunoglobulins, was increased in Hodgkin's disease and decreased in lymphocytic lymphoma. Cellular immune defects in all tests were evident in patients with widespread (localized or generalized) disease. It is suggested that these immune responses, as indices of widespread disease, could be taken into account to increase the accuracy of the staging of various lymphomas. (18 refs.)

- 77-2086 **Control of Immunity in Parental and F<sub>1</sub> Hybrid Mouse Strains to a Spontaneously Arising Parental Tumor 1. Correlation Between In Vitro and In Vivo Anti-Tumor Reactivity in Responsive and Unresponsive Animals.** (Eng.) Levy, R. B. (Immunology Branch, NCI, NIH, Bethesda, MD) Shearer, G. M.; Waksal, S. D. *Eur J Immunol* 7(3): 170-174; 1977.

The in vivo and in vitro cellular immunity of AKR/J, AKD<sub>2</sub>F<sub>1</sub>, (AKR/J x C57BL/6) F<sub>1</sub>, AKB6F<sub>1</sub>, and (AKR/J x DBA/2) F<sub>1</sub> mouse strains to a spontaneously arising murine tumor is examined. AKR carcinomalike tumor cells were cultured both in vitro and in vivo. To compare the ability of AKR/J, (AKD<sub>2</sub>)F<sub>1</sub>, and (AKB6)F<sub>1</sub> mice to respond to the parental tumor in vivo, groups of animals were injected sc with  $5 \times 10^6$  cells, and growth and regression of the tumors were noted at various times. The AKR/J parent mice rejected this dosage as did the (AKB6) F<sub>1</sub> mice. The (AKD<sub>2</sub>)F<sub>1</sub> mice, in contrast, developed progressive lesions. Sensitization cultures containing AKR/J spleen cells and irradiated AKR/J carcinomalike cells were incubated and specific antitumor effector cells harvested following a 5-day incubation period. The primary in vitro cell-mediated immune responses paralleled the in vivo responses in the three strains observed. The generation of suppressor (antitumor effector) cell populations appears to correlate with the strength or weakness of the antitumor response in the animals tested. (22 refs.)

- 77-2087 **Inflammatory Cells in Solid Murine Neoplasms. III. Cytotoxicity Mediated In Vitro by Macrophages Recovered from Disaggregated Regressing Moloney Sarcomas.** (Eng.) Russell, S. W. (Dept. Immunopathology, Scripps Clinic and Res. Foundation, 10666 N. Torrey Pines Road, La Jolla, CA 92037) Gillespie, G. Y.; McIntosh, T. *J Immunol* 118(5): 1574-1579; 1977.

The cytotoxic properties of macrophages recovered from a tumor successfully rejected by its host were characterized. The macrophages were recovered from disaggregated, spon-

taneously regressing Moloney sarcomas for in vitro testing of their cytotoxic capabilities. A heat-stable, soluble, dialyzable inhibitor of thymidine incorporation was produced by the macrophages. As measured by release of <sup>3</sup>Cr, macrophages from tumors efficiently lysed Bd-1 lymphoma cells at a ratio of three macrophages to one target cell. Most of the cytolytic potential was lost within 24 hr. When macrophages were replated at high cell densities, killing was restored. Production of the inhibitor of thymidine incorporation in vitro decreased with time. In contrast to the singularly abrupt and extensive loss in cytolytic activity, the amounts of soluble inhibitor in supernatants decreased gradually over the first 3 days macrophages were in culture. Macrophages recovered from regressing sarcomas are cytolytic, but, unlike T lymphocytes, killing by these inflammatory cells is antigenically nonspecific. (26 refs.)

- 77-2088 **Immunological Studies of Patients with Asbestosis. I. Studies of Cell-mediated Immunity.** (Eng.) Kagan, E. (American Natl. Red Cross Blood Program, Natl. Headquarters, Washington, DC 20006) Solomon, A.; Cochrane, J. C.; Beissner, E. I.; Gluckman, J.; Rocks, P. H.; Webster, I. *Cell Exp Immunol* 28: 261-267; 1977.

A variety of cancers have been documented in patients exposed to asbestos dust. Since a deranged immune system may play a role in cancer development, the general level of immunocompetence was studied in 26 patients with radiographically defined asbestosis who might be at risk of developing asbestos-related neoplasms. Statistical comparisons were made with a comparable control group. A disproportionate number of the patients displayed cutaneous anergy to certain recall antigens and to 2,4-dinitrochlorobenzene. In vitro studies of cellular immunity, as evaluated by phytohemagglutinin-induced proliferative and cytotoxicity assays, showed significantly lower values among the patient group. Serum inhibitors of mitogen-induced lymphocyte transformation were also detected in several patients. The possible significance of these findings is discussed. The presumed carcinogenic properties of asbestos dust may act in concert with a defective immune apparatus in asbestosis patients, with the subsequent development of malignancy occurring as a result of these phenomena. (38 refs.)

- 77-2089 **Cellular Immunity to the Mammary Tumour Virus in Mice Bearing Primary Mammary Tumours.** (Eng.) Creemers, P. (Radiobiological Inst. TNO, Lange Kleiweg 151, Rijswijk ZH, Netherlands) Bentvelzen, P. *Eur J Cancer* 13(6): 503-510; 1977.

The development of cellular immunity against murine mammary tumor virus (MTV) during the growth of primary mammary tumor in BALB/c, BALB/cfC3H, and GR mice was studied by measuring WBC stimulation and WBC adherence

inhibition in the presence of purified MTV. WBC react against group-specific antigens of different MTV strains in these assays. Purified Rauscher murine leukemia virus was used as a control. In both assays, MTV-specific reactivity peaked at a tumor wt of about 1 g; afterward, it almost completely disappeared. It increased again to about half of the original level when the tumor reached a wt of about 3 g. Peak blastogenesis of lymph node cells was observed at a tumor wt somewhat higher than that when the peak blastogenesis of spleen cells occurred. The general decrease in antiviral cellular immunity during tumor growth may have been due to the saturation of activated WBC with viral antigens or to the occurrence of suppressor cells. A cyclic activity of macrophages in the processing of viral antigens may have caused the fluctuations in cellular responsiveness. (32 refs.)

specific antitumor antibody (IgG-2a subclass) by absorption with T1699 cells abolished the ADCC activity at the level of the target cell coating, but did not alter the level of effector-arming activity. Fractionation on Sephadex G-20 indicated that there is more than one factor capable of inducing the specific monocyte-mediated lysis of target cells in vitro, including immune complexes, free IgG-2a 7S antibody (ADCC), and another factor(s) with a molecular wt very close to that of 7S immunoglobulins. (24 refs.)

- 77-2090 **Immunologic Responses to a Murine Mammary Adenocarcinoma. I. Passive Transfer of Immunity by Sera from Tumor-bearing Mice.** (Eng.) Yamamura, Y. (Dept. Basic and Clinical Immunology and Microbiology, Medical Univ. South Carolina, Charleston, SC 29401) Virella, G.; Haskill, J. S. *Int J Cancer* 19(5): 707-716; 1977.

DBA/2 mice bearing a syngeneic mammary adenocarcinoma (T1699) produced high levels of tumor-specific antibody that were detected by indirect immunofluorescence and subsequently identified as the IgG-2a subclass. Tumor-bearer sera passively administered to normal recipients (0.4 or 0.5 ml ip) protected the animals from subsequent challenge with T1699 tumor cells but not from challenge with a non-cross-reacting syngeneic tumor, SaD, fibrosarcoma ( $10^5$  cells sc). Administration of sera prior to tumor challenge was found to be more effective than treatment after the challenge. The protective effect of the sera appeared to parallel both antibody titers and the appearance of concomitant immunity. However, sera absorbed with T1699 cells, with the indirect fluorescent antibody titers reduced more than a hundredfold, conferred an almost identical level of protection. Immune suppression of serum recipients before serum transfer abolished the effect, suggesting that protection depended on a cellular immune response by the host in addition to the possible protective effect(s) of humoral antibody. (36 refs.)

- 77-2091 **Immunologic Responses to a Murine Mammary Adenocarcinoma. II. Monocyte Effector Activation by Humoral Factors.** (Eng.) Yamamura, Y. (Dept. Basic and Clinical Immunology and Microbiology, Medical Univ. South Carolina, Charleston, SC 29401) *Int J Cancer* 19(5): 717-724; 1977.

Sera from DBA/2 mice bearing a syngeneic mammary adenocarcinoma (T1699) not only induced specific antibody-dependent cellular cytotoxicity (ADCC) mediated by monocytes (and/or macrophages), but also armed (activated) these effector cells into specific killer cells in vitro. Removal of

- 77-2092 **The Splenic Influence on BCG-mediated Therapy in a Syngeneic Guinea Pig Tumor Model.** (Eng.) Jessup, J. M.; Brandhorst, J. S.; Peters, L. C.; Hanna, M. G. In: *Immuno-aspects of the Spleen. Proceedings of a Conference on Immunodynamics held in Cleveland, Ohio, May 19-20, 1976.* Battisto, J. R.; Streilein, J. W., eds. (New York: North-Holland Publishing Co.): pp. 391-398; 1976.

The effects of splenectomy on the line-10 hepatocarcinoma tumor model and on BCG-mediated tumor regression were studied in inbred male guinea pigs. The animals were splenectomized at different times before and after tumor inoculation to determine the role of the spleen during different phases of the immune response to a weakly antigenic tumor. There was no difference in the growth rate of tumors between splenectomized weanlings and sham-operated controls or between 3-mo-old guinea pigs splenectomized 4 days after tumor inoculation and sham-operated animals. Guinea pigs treated with BCG (diluted 1:100) and splenectomized, guinea pigs treated with diluted BCG only, and untreated guinea pigs demonstrated no differences in tumor growth. When splenectomy was performed 11 days after tumor inoculation without BCG treatment, tumor growth was significantly increased compared to untreated controls and guinea pigs receiving BCG. Two months after weanling guinea pigs were splenectomized, they and their unoperated littermates were inoculated iv with line-10 cells. These animals were then given either BCG or BCG admixed with  $10^7$  irradiated tumor cells iv 1 day later. Booster shots of  $10^7$  irradiated cells were administered 7 days after tumor inoculation. The survival rate was better in all four vaccinated groups than in untreated controls. The splenectomized guinea pigs generally survived longer than their unoperated littermates who received the same initial vaccination treatment. Splenectomy does not inhibit the growth of weakly immunogenic tumors. (22 refs.)

- 77-2093 **Natural Immunity to Endogenous Oncornaviruses in Mice.** (Eng.) Ihle, J. N. (Basic Res. Program, NCI, Frederick, MD 21701) Hanna, M. G. *Contemp Top Immunobiol* 6: 169-194; 1977.

Studies of the natural humoral immune response to endogenous C-type viruses in mice are summarized, and interpretations regarding the functional role of autogenous immunity to endogenous murine leukemia viruses (MuLV) of AKR



mice are discussed. The findings indicate that mice are not immunologically tolerant to the expression of endogenous C-type viruses, but rather maintain a state of immunological surveillance. Although these viruses are endogenous and they are transmitted vertically, the host recognizes them as infectious agents. The lack of immunological tolerance provides a basis for the immunological regulation of virus expression and the control of pathogenesis. These experiments also suggest that the immune regulation of virus-mediated pathogenesis could be expected to contribute significantly only in a relatively restricted set of circumstances of intermediate levels of expression, its influence being modified by other genetic factors involved with expression and infectivity. (49 refs.)

- 77-2094 **Cyclic AMP and Immune Responses: The Role of Polyadenylic: Polyuridylic Acid.** (Eng.) Plescia, O. J. In: *Modulation of Host Immune Resistance in the Prevention or Treatment of Induced Neoplasias*. NCI and John E. Fogarty International Center for Advanced Study in the Health Sciences. (Bethesda, Maryland): pp. 163-168; 1974.

Evidence is presented that the immune response of mice to sheep RBC is associated with changes in the splenic level of cyclic AMP (cAMP) that are linked to initiation of antibody formation and proliferation of antibody-forming cells. It is also shown that polyadenylic: polyuridylic acid [poly(A:U)] acts synergistically with sheep RBC to elevate the level of endogenous cAMP. Stimulation occurs during the initiation phase of the immune response, and it requires the presence of antigen. Because amounts in excess of optimum give decreased stimulation, the amount of poly(A:U) administered is a factor. cAMP increases during the initiation phase, returns to base level, and then decreases during the proliferation phase. Consequently, depending on the time of its administration relative to antigen, poly(A:U) is both immunosuppressive and immunostimulating. With knowledge of the transient and biphasic character of the cAMP response to antigen, there is a rational basis for exploiting drugs that act synergistically with antigen in stimulating endogenous cAMP as a means of enhancing or suppressing the immune response. Poly(A:U) is one such agent. (12 refs.)

- 77-2095 **Development of Spontaneously Metastasizing Mammary Carcinomas (MT) as the Consequence of Host Immune Surveillance During Chemical Carcinogenesis (Meeting Abstract).** (Eng.) Kim, U. (Dept. Pathology, Roswell Park Memorial Inst., Buffalo, NY 14263) *Fed Proc* 36(3): 1222; 1977. (no refs.)

- 77-2096 **Comparative Analysis of the Tumor Associated Cellular Response to Two Syngeneic Passaged Chemically Induced Murine Fibrosarcomas (Meeting Abstract).** (Eng.) Wood, G. W. (Univ. Kansas Medical Center,

Kansas City, KS 66103) Gollahon, K. A. *Fed Proc* 36(3): 1222; 1977. (no refs.)

- 77-2097 **The Timing of Appearance of Host Response in Murine Tumors (Meeting Abstract).** (Eng.) Karakousis, C. P. (Roswell Park Memorial Inst., Buffalo, NY 14263) Douglass, H. O.; Paolini, N. S.; Goldrosen, M. P. *Fed Proc* 36(3): 1205; 1977. (no refs.)

- 77-2098 **Resistance of Rat Fetal tissues in Utero to Invasion by Maternal Malignant Lymphoma (Meeting Abstract).** (Eng.) McCreary, P. A. (Rush-Presbyterian St Luke's Medical Center, Chicago, IL 60612) Laing, G. H. Hass, G. M. *Fed Proc* 36(3): 1243; 1977. (no refs.)

- 77-2099 **Induction of Tumor Immunity by Conjoint Use of Attenuated Tumor Cells and Glucan (Meeting Abstract).** (Eng.) Luzio, N. R. (Dept. Physiology, Tulane Univ. Sch. Medicine, New Orleans, LA 70112) Browder, W. McNamee, R. *Fed Proc* 36(3): 1242; 1977. (no refs.)

- 77-2100 **The Role of Eosinophils in the Mechanism of Schistosome Immunity (Meeting Abstract).** (Eng.) Li Hsu, S. Y. (Univ. Iowa, Iowa City, IA 52242) Hsu, H. F.; Isacson, P. *Fed Proc* 36(3): 1057; 1977. (no refs.)

- 77-2101 **A Role for Thymocytes in BCG-Induced Resistance to *Schistosoma mansoni* Infection (Meeting Abstract).** (Eng.) Civil, R. H. (Dept. Pathology, Dept. Medicine, Case Western Reserve Univ. and Univ. Hosps. Cleveland, OH 44106) Mahmoud, A. F. *Fed Proc* 36(3): 1057; 1977. (no refs.)

- 77-2102 **Effects of Pretreatment with Formalinized Syngeneic and Allogeneic Rous Sarcoma Cells on Growth of RSV-Induced Tumors in Chickens (Meeting Abstract).** (Eng.) Schierman, L. W. (New York Medical Coll Valhalla, NY 10595) McBride, R. A. *Fed Proc* 36(3): 1243; 1977. (no refs.)

- 77-2103 **Immunity to Syngeneic Lymphoma Cells Raised by Immunization with Allogeneic Lymphoma Cells (Meeting Abstract).** (Eng.) Gillette, R. W. (Cancer Center Hawaii, Univ. Hawaii, Honolulu, HI 96822) Wunderlich, D. A. *Fed Proc* 36(3): 1290; 1977. (no refs.)

- 77-2104 **The Variable Effect of Soluble Tumor Antigen Immunization upon the Growth of Morris Hepatomas (Meeting Abstract).** (Eng.) Tracey, R. S. (V.A. Hosp., San Diego, CA 92161) Wepsic, H. T. *Fed Proc* 36(3): 1243; 1977. (no refs.)
- 77-2105 **Effect of Pertussis Vaccine on the Induction of Tumors in Mice by SV40 Virus (Meeting Abstract)** (Eng.) Hargis, B. J. (Sidney Farber Cancer Inst., Harvard Medical Sch., Boston, MA 02115) Malkiel, S. *Fed Proc* 36(3): 1242; 1977. (no refs.)
- 77-2106 **Suppression of Methyl Cholanthrene (MCA) Induced Fibrosarcoma in Balb/C Mice by Group A Streptococci (Meeting Abstract).** (Eng.) Bhatnagar, R. M. (Rockefeller Univ., New York, NY 10021) Rausen, A. R.; Zabriskie, J. B. *Fed Proc* 36(3): 1242; 1977. (no refs.)
- 77-2107 **Anti-Tumor Activity of *Listeria Monocytogenes* in Strain 13 Guinea Pigs (Meeting Abstract).** (Eng.) Dustoor, M. M. (Univ. Wisconsin, Madison, WI 53706) Blazkovec, A. *Fed Proc* 36(3): 1291; 1977. (no refs.)
- 77-2108 **Relative Effectiveness of Methods for Immunizing Mice to Myeloid Leukemia (Meeting Abstract).** (Eng.) Urnovitz, H. B. (Univ. Michigan, Ann Arbor, MI 48109) Murphy, W. H. *Fed Proc* 36(3): 1243; 1977. (no refs.)
- 77-2109 **Importance of Route and Vaccination-Challenge Interval for Effectiveness of Immunization Against a Mouse Lymphoma (Meeting Abstract).** (Eng.) Kendall, C. (Dept. Zoology, Univ. Texas, Austin, TX 78712) *Fed Proc* 36(3): 1291; 1977. (no refs.)
- 77-2110 **Protection Against Friend Leukemia Virus (FLV) in Mice Treated with Heterologous Anti-gp71 Antisera (Meeting Abstract).** (Eng.) Sanfilippo, F. (Duke Univ. Medical Center, Durham, NC 27710) Lynn, T.; Metzgar, R.; Bolognesi, D.; Collins, J. *Fed Proc* 36(3): 1084; 1977. (no refs.)
- 77-2111 **The Cell Mediated-Focus Reduction Assay for Avian Oncornaviruses (Meeting Abstract).** (Eng.) Whiteaker, R. S. (Dept. Veterinary Microbiology, Univ. Missouri, Columbia, MO 65201) Adldinger, H. K.; Loan, R. W. *Fed Proc* 36(3): 1261; 1977. (no refs.)
- 77-2112 **Cellular Requirements for Expression of Tumor Immunity in Chickens (Meeting Abstract).** (Eng.) Palladino, M. A. (Dept. Pathology, New York Univ. Sch. Medicine, New York, NY 10016) Thorbecke, G. J. *Fed Proc* 36(3): 1222; 1977. (no refs.)
- 77-2113 **Transfer of Age-Related Resistance to Marek's Disease (JMV) by Spleen Cells (Meeting Abstract).** (Eng.) Lam, K. M. (Dept. Microbiology, Temple Univ. Sch. Medicine, Philadelphia, PA 19140) Pasternak, R. D.; Linna, T. J. *Fed Proc* 36(3): 1247; 1977. (no refs.)
- 77-2114 **Arginine Inhibits a Viral Tumor (Meeting Abstract).** (Eng.) Critselis, A. N. (Albert Einstein Coll. Medicine, Bronx, NY 10461) Rettura, G.; Barbul, A.; Seifter, E. *Fed Proc* 36(3): 1163; 1977. (no refs.)
- 77-2115 **Cell-Mediated Immunity to Hamster Type C Virus and Lymphoma in Hamsters (Meeting Abstract).** (Eng.) Datta, S. K. (Baylor Coll. Medicine, Houston, TX 77030) McCormick, K. J.; Trentin, J. J. *Fed Proc* 36(3): 1261; 1977. (no refs.)
- 77-2116 **Characteristics of Human Cell-Associated Immunity to Herpes Simplex Virus, Type 1 (HSV-1) (Meeting Abstract).** (Eng.) Pass, M. K. (NIH, Baltimore Cancer Res. Center, Baltimore, MD 21211) Joseph, J. M.; Mardiney, M. R. *Fed Proc* 36(3): 1228; 1977. (no refs.)
- 77-2117 **Regulation of the Cellular Immune Response to Herpes Simplex Virus (HSV) Antigens by Rabbit and B Lymphocytes (Meeting Abstract).** (Eng.) Meyers, R. L. (Jules Stein Eye Inst., UCLA Sch. Medicine, Los Angeles, CA 90024) Chitjian, P. A. *Fed Proc* 36(3): 1249; 1977. (no refs.)
- 77-2118 **The Immune Behavior Induced by the Vx2 Tumor in Rabbits Characterized at the Major Histocompatibility and Blood Group Loci (Meeting Abstract).** (Eng.) Lancki, D. W. (Univ. Illinois Medical Center, Chicago, IL 60680) Tissot, R. G.; Cohen, C. *Fed Proc* 36(3): 1292; 1977. (no refs.)



- 77-2119 Resistance of Irradiated Mice to Lymphoma Transplants: Importance of the Hh-1/D Region of the H-2 Complex and Preferential Expression in the Spleen.** (Eng.) Cudkowicz, G.; Riccardi, C. In: *Immunodynamics of the Spleen. Proceedings of a Conference on Immunodynamics, Cleveland, Ohio, May 19 and 20, 1976*. Battisto, J. R.; Streilein, J. W., eds. (New York: North-Holland Publishing Co.): pp. 373-375; 1976.

The genetic differences responsible for the localized resistance of irradiated mice to lymphoma transplants were found to reside in the Hh-1/D region of the H-2 complex. Differences at other regions were inconsequential for lymphoma growth in irradiated hosts. The administration of silica particles rapidly abrogated resistance in both euthymic and athymic animals. An in vitro model of resistance was developed in which F<sub>1</sub> lymphocytes generated cell-mediated cytotoxic activity specific for parental spleen and lymphoma cells. The results of these in vitro and in vivo experiments pointed to a resemblance to hemopoietic histocompatibility (the rejection of foreign bone marrow grafts by irradiated mice), and they indicated that the products of Hh genes are expressed in lymphoma cells. Mechanisms of anti-Hh responses may be of significance in tumor immunity. (no refs.)

- 77-2120 Influence of H-2 Complex on Susceptibility to Infection by Murine Leukemia Virus.** (Eng.) Tucker, H. S. (Hematology Service, New England Medical Center Hosp., Boston, MA 02111) Weens, J.; Tsiichlis, P.; Schwartz, R. S.; Khiroya, R.; Donnelly, J. *J Immunol* 118(4): 1239-1243; 1977.

The effect of the H-2 complex on the early phase of infection by murine leukemia virus (B-tropic MuLV) was studied. Several strains of congenic resistant mice were used. In mice differing only in H-2 haplotype, a gene(s) within the H-2 complex was found that determined a high (H-2k, H-2d, H-2a) or low virus titer (H-2b, H-2q). For example, 10 wk after infection with virus (200 plaque-forming units), B10 mice had significantly lower titers of infectious virus in their spleens than B10.A mice. (B10 × B10.A)<sub>F1</sub> mice were phenotypically like the B10.A parent; thus, susceptibility to high virus titers was inherited as a dominant trait. Kinetics experiments with strains B10 and B10.A showed that no infectious virus was detectable in both until 4 wk after infection. From 4 to 6 wk the titers of infectious virus rose, but from 6 to 10 wk, the titers in B10 mice declined progressively. This suggests that there is a mechanism in B10, lacking in B10.A, that restricts viral replication after infection. Antibody to viral envelope antigens titers differed significantly between the two strains, but the antibody did not appear to be involved in virus elimination. Genes outside the H-2 complex influenced MuLV titers after infection as well. (20 refs.)

- 77-2121 H-2 Compatibility Requirements for Interaction of Friend Virus (FV) Induced T Suppressor**

**Cells with Mitogen Responsive T Cells (MRC) (Meeting Abstract).** (Eng.) Kumar, V. (Dept. Pathology, Boston Univ. Sch. Medicine, Boston, MA 02118) Bennett, M. *Fed Proc* 36(3): 1202; 1977. (no refs.)

- 77-2122 Induction of T-Helper and T-Suppressor Activities with Thymic Extracts (Meeting Abstract).** (Eng.) Rule, A. H. (Boston Coll., MA 02167) Miller, H. *Fed Proc* 36(3): 1183; 1977. (no refs.)

- 77-2123 Identification of Splenic Suppressor Cell Activity in Mice with MuMTV-Induced Mammary Tumors (Meeting Abstract).** (Eng.) Rudczynski, A. B. (Dept. Biology, Michigan Cancer Foundation, Detroit, MI 48201) Mortensen, R. F. *Fed Proc* 36(3): 1222; 1977. (no refs.)

- 77-2124 The Relationship Between Tumor Immunogenicity and the Appearance of Splenic Immunosuppressor Cells in Tumor Bearing Mice (Meeting Abstract).** (Eng.) Altman, A. (Lab. Immunobiology, NCI, NIH, Bethesda, MD 20014) Bast, B.; Bast, R. C.; Rapp, H. J. *Fed Proc* 36(3): 1274; 1977. (no refs.)

- 77-2125 Role of Suppressor Cells in Enhanced Tumor Growth Caused by Anti-Th-B Antibodies (Meeting Abstract).** (Eng.) Kakimoto, K. (Roswell Park Memorial Inst., Buffalo, NY 14263) Fuji, H.; Grossberg, A. L.; Pressman, D. *Fed Proc* 36(3): 1290; 1977. (no refs.)

- 77-2126 Suppressor Activity in the Spleen of Tumor Allosensitized Mice (Meeting Abstract).** (Eng.) Argyris, B. F. (Dept. Microbiology, Upstate Medical Center, Syracuse, NY 13210) *Fed Proc* 36(3): 1274; 1977. (no refs.)

- 77-2127 Soluble Suppressor Factor from the Spleens of Tumor Bearing Mice (Meeting Abstract).** (Eng.) Subramanian, C. (Univ. Minnesota, Minneapolis, MN 55455) Yu, S.; McKhann, C. F. *Fed Proc* 36(3): 1274; 1977. (no refs.)

- 77-2128 Generation of Suppressor Cells in Mice Immunized with M Locus-incompatible Lymphocytes.** (Eng.) Matossian-Rogers, A. (Tissue Immunology Unit, London Hosp. Medical Coll., London, E1.4D, England) Festenstein, H. *Transplantation* 23(4): 316-321; 1977.

The effect of alloimmunization of mice with M locus-incompatible lymphocytes on the generation of suppressor cells in the immunized host was studied. The lymph node cells from these alloimmunized mice decreased the in vitro cytotoxic response of normal cells to H-2 alloantigens. M locus-preimmunized lymphocytes suppressed the generation of effectors by normal CBA/H cells. This effect was not simply attributable to dilution of the effector cell population with cells unable to develop cytotoxicity. Injection of M locus-preimmunized cells into the footpads of  $F_1$  hybrid mice decreased popliteal lymph node enlargement. The restimulation of suppressor cells in culture with the specific M locus was necessary for suppression of the effector cell generation. The suppressive effect was demonstrated to be attributable to M locus determinants utilizing M locus-pseudocongenic mice. The investigation of inhibitory effects on T-cell cytotoxicity due to serologically undetectable lymphocyte-activating determinants may lead to an elucidation of suppressive mechanisms permitting syngeneic tumor growth. (22 refs.)

77-2129 **Differential Tumor Immunogenicity of L1210 and its Sublines. An Inverse Relationship Between Immunogenicity and an Ability to Elicit Splenic "Suppressor" Activity (Meeting Abstract).** (Eng.) Fuji, H. (Roswell Park Memorial Inst., Buffalo, NY 14263) Pressman, D. *Fed Proc* 36(3): 1204; 1977. (no refs.)

77-2130 **Inhibition of H-2 Directed, T-Cell Mediated Cytolysis of Mouse Target Cells by Sugar Derivatives.** (Eng.) Trefts, P. E.; Alhadeff, J. A. In: *Oncodevelopmental Gene Expression. Papers Presented at a Conference Sponsored by the International Research Group for Carcinoembryonic Proteins, and Held at San Diego, May 29-June 1, 1976.* International Research Group for Carcinoembryonic Proteins. (San Diego, California): pp. 517-522; 1976.

A series of disaccharide analogs, or nitrophenyl sugars, were examined as potential inhibitors of cytotoxic T-cell killing of H-2 defined target cells. The experiments were performed in C57BL/6 (B/6, H-2b), C57BL/10 (B10, H-2b), B10.D2/nSn (H-2d), B10.A(5R) (H-2b-d) and DBA/2 (H-2d) mice; the inhibitory potency of all compounds was determined at a single final concentration of approx 5 mM. P-nitrophenyl- $\alpha$ -fucose routinely inhibited T-cell killing of EL-4 (B/6) and B15 (DBA/2) targets in several allogeneic effector combinations. Less marked inhibition was obtained with  $\beta$ -L-fucose and  $\alpha$ -D-mannose compounds. The  $\alpha$ -L-fucose derivative reduced effector activity over 27-fold in the combination B/6 anti-P815 and 22-fold in the reverse combination. In the former combination,  $\alpha$ -D-mannose appears to inhibit more killing than the  $\beta$ -L-fucose compound; the reverse was observed in the DBA/2-EL-4 situation. Primed B10 responder spleens were stimulated in culture with B10.D2 lymphocytes and treated against B10.A(5R)LPS blast targets. The  $\alpha$ -

L-fucose derivative substantially reduced T-cell effector activity against either K or D end targets. The  $\alpha$ -D-mannose and  $\beta$ -L-fucose compounds were less inhibitory, and the inhibition was concentration-dependent. Other haplotype combinations gave similar results. These derivatives neither increased nor decreased the spontaneous release of tumor and blast targets. (10 refs.)

77-2131 **Cytolytic Efficiency of Isolated Primary and Secondary Lymphocytes (Meeting Abstract).** (Eng.) Grimm, E. (Dept. Microbiology and Immunology, UCLA, Los Angeles, CA 90024) *Fed Proc* 36(3): 1280; 1977. (no refs.)

77-2132 **Role of Aerobic and Anaerobic Energy Metabolism in T-Cell Mediated Cytolysis (Meeting Abstract).** (Eng.) MacDonald, H. R. (Ontario Cancer Foundation, London, Ontario N6A 4G5, Canada) Koch, C. J. *Fed Proc* 36(3): 1324; 1977. (no refs.)

77-2133 **Inhibition of T Cell-Mediated Cytolysis In Vitro with Unlabeled Target Cells and with Subcellular Alloantigen Preparations (Meeting Abstract).** (Eng.) Linna, T. J. (Dept. Immunology, Swiss Inst. for Experimental Cancer Res., Lausanne, Switzerland,) Cerottini, J. C.; Brunner, K. T. *Fed Proc* 36(3): 1325; 1977. (no refs.)

77-2134 **Studies on the Specific Inhibition of Cell-mediated Cytolysis In Vitro by Unlabeled Cells and by Particulate Alloantigens.** (Eng.) Linna, T. J.; Nobis, P.; Cerottini, J. C.; Brunner, K. T. In: *Immuno-aspects of the Spleen. Proceedings of a Conference on Immunodynamics, Cleveland, Ohio, May 19 and 20, 1976.* Battisto, J. R.; Streilein, J. W., eds. (New York: North-Holland Publishing Co.): pp. 401-411; 1976.

The inhibition of cell-mediated cytotoxicity was assessed. Alloantigen preparations obtained from RB1-5 (C57B1/6) and LSTRA (BALB/c) lymphoma cells were capable of inducing the secondary formation of cytolytic T lymphocytes (CTL). The number of lytic units per  $10^6$  viable cells recovered or per culture in a 3.5-hr standard CTL assay was of the same order of magnitude as when the corresponding allogeneic cells were utilized to generate CTL. When cytochalasin B was added after 15 min incubation at 37 C, the cytolytic activity of the cell populations was much lower. When a constant number of alloimmune BALB/c (H-2d) lymphoid cells [restimulated in vitro with H-2b (RB1-5) alloantigen] was preincubated with unlabeled RB1-5 cells before the addition of



<sup>51</sup>Cr-labeled RB1-5 target cells, followed 15 min later by cytochalasin B, cytolytic activity was inhibited significantly. Inhibition was found when unlabeled LSTRA (H-2d) cells were used to inhibit alloimmune C57Bl/6 cells restimulated in vitro with H-2d (LSTRA) antigen. This antigen was able to inhibit specifically the cytolysis by C57Bl/6 (H-2b) lymphocytes restimulated with the H-2d antigen preparation, and it had very little activity in the control system. Although alloantigen preparations are able to induce CTL formation regularly, only suggestive evidence is obtained for their ability to inhibit cell-mediated cytolysis. (16 refs.)

77-2135 **Tumor-Host Interaction in Relation to Immune Complexes and Carcinoembryonic Antigen Levels (Meeting Abstract).** (Eng.) Daugharty, H. (Center for Disease Control, Atlanta, GA 30333) Strobel, P. L.; Hicklin, M. D. *Fed Proc* 36(3): 1213; 1977. (no refs.)

77-2136 **Circulating Immune Complexes in Syngeneic and Allogeneic Rats Bearing Moloney Sarcomas (Meeting Abstract).** (Eng.) Jennette, J. C. (Scripps Clinic and Res. Foundation, La Jolla, CA 92037) Feldman, J. D. *Fed Proc* 36(3): 1204; 1977. (no refs.)

77-2137 **Treatment of Leukemic Mice with Syngeneic Hybrid Cells: Activation of Specific Cellular Immunity (Meeting Abstract).** (Eng.) Liang, W. (La Rabida-Univ. Chicago Inst., Chicago, IL 60649) Cohen, E. P. *Fed Proc* 36(3): 1243; 1977. (no refs.)

77-2138 **In Vitro Generation and Adoptive Transfer of Cellular Immunity to a Syngeneic Tumor (Meeting Abstract).** (Eng.) Moe, M. (Univ. Minnesota, Minneapolis, MN 55455) Simmons, R. L. *Fed Proc* 36(3): 1205; 1977. (no refs.)

77-2139 **Destruction of Syngeneic Tumors as "Innocent Bystanders": Comparison of Mouse and Guinea Pig (Meeting Abstract).** (Eng.) Berkelhammer, J. (Basic Res. Program, NCI Frederick Cancer Res. Center, Frederick, MD 21701) Kripke, M. L.; Caines, S.; Hanna, M. G. *Fed Proc* 36(3): 1223; 1977. (no refs.)

77-2140 **Tumor Growth Inhibition Mediated by Lymphocytes Sensitized In Vitro to a Syngeneic Murine Teratocarcinoma 402AX (Meeting Abstract).** (Eng.) Bartlett, P. F. (The Johns Hopkins Univ., Baltimore, MD

21218) Fenderson, B. A.; Edidin, M. *Fed Proc* 36(3): 1290; 1977. (no refs.)

77-2141 **Role of T and B Interaction in Tumor Immunity (Meeting Abstract).** (Eng.) Marusic, M. (UT-Oak Ridge Graduate Sch. Biomedical Science and Biology Div., ORNL, 2 Oak Ridge, TN 37830) Goodman, J. W.; Shinpock, S. G. *Fed Proc* 36(3): 1205; 1977. (no refs.)

77-2142 **Effect of T Lymphocytes on In Vivo Growth of a Syngeneic Moloney Sarcoma (BM2) in Brown Norway (BN) Rats (Meeting Abstract).** (Eng.) Halliburton, B. L. (Scripps Clinic and Res. Foundation, La Jolla, CA 92037) *Fed Proc* 36(3): 1290; 1977. (no refs.)

77-2143 **Specificity of T Cell Immunity to a BALB/c Myeloma Tumor (Meeting Abstract).** (Eng.) Russell, J. H. (Center Cancer Res., Massachusetts Inst. Technology, Cambridge, MA 02139) Terres, G.; Eisen, H. N. *Fed Proc* 36(3): 1261; 1977. (no refs.)

77-2144 **Nutritional Effects on Syngeneic Tumor Immunity and Carcinogenesis in Mice (Meeting Abstract).** (Eng.) Syrotuck, J. A. (Univ. Washington, Seattle, WA 98195) Worthington, B. S. *Fed Proc* 36(3): 1163; 1977. (no refs.)

77-2145 **Effects of Intravenous Hyperalimentation (IVH) on Tumor Growth and Host Immunocompetence (Meeting Abstract).** (Eng.) Daly, J. M. (Univ. Texas Medical Sch., Houston, TX 77030) Copeland, E. M.; Guinn, E. *Fed Proc* 36(3): 1163; 1977. (no refs.)

77-2146 **Effect of Malnutrition on Syngeneic Tumor Growth (Meeting Abstract).** (Eng.) Malave, I. (I.V.I.C. Ap 1827, Caracas, Venezuela) Blanca, I. *Fed Proc* 36(3): 1254; 1977. (no refs.)

77-2147 **Nature of Humoral Factors in Syngeneic Mammary Tumor Bearing Rats Blocking Cell-Mediated Cytotoxicity (CMC) (Meeting Abstract).** (Eng.) Huber, S. A. (Stanford Univ. Medical Center, Stanford, CA 94305) Lucas, Z. J. *Fed Proc* 36(3): 1306; 1977. (no refs.)

- 77-2148 **Immunofluorescent Studies on Chimpanzee Humoral Responses to Human Melanoma Cells.** (Eng.) Leong, S. P. (Dept. Surgery, Tulane Univ. Sch. Medicine, 1430 Tulane Ave., New Orleans, LA 70112) Hornung, M. O.; Krementz, E. T. *Oncology* 33(5/6): 246-249; 1976.

Surface antigens were demonstrated in five human melanoma cell lines by indirect immunofluorescence. Seven of eight chimpanzees immunized with melanoma cells failed to produce antibody to any of these lines, but the serum of one of the chimpanzees reacted positively with four lines. Membrane fluorescence but no cytoplasmic or nuclear fluorescence was shown. Mechanically removed cells gave bright full-ring fluorescence, but trypsinized cells demonstrated patchy or aggregated surface fluorescence. Negative serum controls were normal human serum, normal chimpanzee serum, and saline. Negative cell controls were human fibroblasts, WI-38 cells, HeLa cells, monkey kidney cells, and human kidney cells. When the antiserum of the chimpanzee was absorbed with the normal skin fibroblasts of one patient, it reacted positively with her melanoma cells. However, chimpanzee antiserum absorbed with these melanoma cells lost the ability to react with the other melanoma cells. The reactive immunoglobulin (Ig) was C'-fixing IgG, as shown by a complement-tagging experiment carried out on one melanoma cell line. It was concluded that the immunized chimpanzee produced antibody to surface antigen(s) common to four of the melanoma cell lines tested. (25 refs.)

- 77-2149 **The Development of a Cytotoxic Cellular Immune Response to the MKS-TU5 Tumor in Mice with Growing Tumors (Meeting Abstract).** (Eng.) Meyer, A. A. (Univ. Chicago, Chicago, IL 60637) Hunter, R. L.; Wissler, R. W. *Fed Proc* 36(3): 1205; 1977. (no refs.)

- 77-2150 **Localized Suppression of Cytotoxic Lymphocyte Responses (Meeting Abstract).** (Eng.) Peavy, D. L. (Dept. Pathology, Harvard Medical Sch., Boston, MA 02115) Pierce, C. W. *Fed Proc* 36(3): 1183; 1977. (no refs.)

- 77-2151 **T Lymphocyte Induction of Non-T Cell-mediated Nonspecific Cytotoxicity. I. Introduction Mechanisms.** (Eng.) Mackler, B. F. (Immunology Lab., Dental Sciences Inst., Post Office Box 20068, Houston, TX 7025) O'Neill, P. A.; Meistrich, M. *Eur J Immunol* 7(2): 5-61; 1977.

The cytotoxicity of human mononuclear cells and subpopulations from normal healthy donors was assessed in direct microcytotoxicity assays using autologous (gingival fibroblasts) and allogeneic (human melanoma line SH-1 and lung fibroblast line WI-38) target cells. The rosetting of human T cells with sheep erythrocytes (E) caused the release of a soluble

factor(s) termed the E-rosetting supernatant (ERSup), which induced quiescent non-T cells to mediate nonspecific cytotoxicity. This was determined by the following results: (1) mononuclear cells incubated with sheep E at 37 C failed to form rosettes or produce ERSup; (2) T cells incubated with other heterologous E were unable to form rosettes or produce ERSup; (3) removal of adherent sheep E from T cells producing ERSup stopped further ERSup release. Control experiments eliminated the participation of fetal calf serum and sheep E eluates and the nonspecific activation of macrophages by physical packing during rosetting. The cytotoxic cells were characterized as non-E-rosetting, nonphagocytic, glass-adherent lymphocytes. The E-rosette-induced release of ERSup does not appear to involve a monocytic cell population. It probably involves the release of some preformed membrane-bound constituent or the activation of a preformed intracellular molecule, rather than de novo protein synthesis. Cell separation procedures (and, possibly, in vivo events) that activate T cells may also induce non-T-cell-mediated nonspecific cytotoxicity. (26 refs.)

- 77-2152 **Relationship of Human Natural Lymphocyte-mediated Cytotoxicity to Cytotoxicity of Breast-Cancer-derived Target Cells.** (Eng.) Cannon, G. B. (Dept. Immunology, Litton Bionetics, Inc., 5516 Nicholson Lane, Kensington, MD 20795) Bonnard, G. D.; Djeu, J.; West, W. H.; Herberman, R. B. *Int J Cancer* 18(4): 487-497; 1977.

Mononuclear cells from 115 individuals were tested in a 4-hr chromium release assay against two breast cancer cell lines, G11 and MCF-7, and a myeloid cell line, K-562, that is sensitive to natural cytotoxicity. The results were analyzed in a manner designed to detect hyperreactivity against the breast cell lines relative to the level of reactivity against K-562. Twelve of 18 breast cancer patients were hyperreactive to G11 and 10/18 to MCF-7. Fibroadenoma patients were similar to normal women, with 0/11 hyperreactive to G11 and 1/11 to MCF-7. In contrast, normal men were hyperreactive to G11 (7/17) but not to MCF-7 (2/17). Colon and lung cancer patients were also more hyperreactive to G11 (4/8 and 4/6, respectively) than to MCF-7 (1/8 and 1/6, respectively). Patients with fibrocystic disease resembled breast cancer patients, with some hyperreactive to both G11 (3/8) and MCF-7 (2/8). The method yielded similar results in another group of subjects: only 1/14 normal women were hyperreactive to G11 compared with 12/17 breast cancer patients. It is concluded that tests against K-562 and MCF-7 can be used to detect patterns of cell-mediated reactions that are characteristic of patients with breast cancer. (28 refs.)

- 77-2153 **Augmentation of Natural Cytotoxic Reactivity of Mouse Lymphoid Cells Against Syngeneic and Allogeneic Target Cells.** (Eng.) Herberman, R. B. (Lab. Immunodiagnosis, NCI, Bethesda, MD 20014) Nunn, M. E.;



Holden, H. T.; Staal, S.; Djeu, J. Y. *Int J Cancer* 19(4): 555-564; 1977.

The possibility that the natural cytotoxic reactivity of mouse lymphoid cells could be augmented by in vivo challenge with tumor cells and other materials was investigated. Ip or sc inoculation of either RBL-5 or YC8 ascitic tumor cells ( $1 \times 10^7$  cells) led to a significant increase in the reactivity of spleen cells of young BALB/c nude mice against syngeneic lymphoma cells. BALB/c thymus cells also augmented spleen cell cytotoxicity. In older mice with low levels of natural cytotoxicity, tumor cell inoculation caused the rapid reappearance of cytotoxicity. This augmented reactivity peaked 3 days after inoculation and then declined rapidly. The specificity of the stimulated reactivity was similar to that seen with natural cell-mediated cytotoxicity; ie, the detected antigens were restricted to mouse tumor cells and thymocytes. After boosting, the effector cells had the same cell surface characteristics as the spontaneously cytotoxic cells; they were nonadherent, nonphagocytic, and only weakly sensitive to treatment with antitheta serum plus complement. The natural cytotoxicity of BALB/c mice was also markedly augmented by murine viruses such as murine sarcoma virus and lymphocytic choriomeningitis virus. These effector cells also had the same properties as the natural cytotoxic effector cells. The ability of tumor cells and viruses to augment natural cytotoxic reactivity should allow a better understanding of the in vivo role of this reactivity in tumor growth resistance. (32 refs.)

**77-2154 In Vitro Reactivity of Splenic Lymphocytes from Normal and UV-irradiated Mice Against Syngeneic UV-induced Tumors.** (Eng.) Fortner, G. W. (Basic Res. Program, NCI Frederick Cancer Res. Center, Post Office Box B, Frederick, MD 21701) Kripke, M. L. *J Immunol* 118(4): 1483-1487; 1977.

The relative antigenicity and cross-reactivity of syngeneic (syn) UV-induced fibrosarcomas in C3H- mice were examined by an in vitro microcytotoxicity test. In addition, the antitumor reactivity of lymphocytes from normal (regressor) and UV-irradiated (progressor) mice following challenge with these tumors was compared. Spleen cells of mice injected sc with  $1 \times 10^6$  syn UV-induced fibrosarcoma cells were highly cytotoxic. Upon removal of an adherent cell population by filtration, the spleen cells reacted with the immunizing tumor, but not with other syn UV-induced tumors, methylcholanthrene (MCA)-induced tumors, or normal C3H-lines. The specific cytotoxicity produced by a syn UV-induced tumor was much higher than that produced by syn MCA-induced tumors; this increased reactivity was attributed to a population of  $\theta$ -antigen-bearing lymphocytes. In further experiments, the cytotoxic reactivity of splenic lymphocytes against syn UV-induced tumors between mice irradiated with UV light for 3 mo was compared against unirradiated controls. After inoculation of the mice with  $1 \times 10^7$  tumor cells, lymphocytes from the unirradiated (regressor) mice showed a high degree of cytotoxicity that reached a max

after 8 days. No reactivity, however, could be detected in the spleens of irradiated (progressor) mice. This suggests that the population of  $\theta$ + cytotoxic effector cells generated in unirradiated mice is absent, reduced considerably, or inhibited in the irradiated animals. (10 refs.)

**77-2155 Specificities in Natural Cell-mediated Cytotoxicity by the Cross-Competition Assay.** (Eng.) Takasugi, M. (Dept. Surgery, Sch. Medicine, Univ. California, Los Angeles, CA 90024) Koide, Y.; Akira, D.; Ransley, A. *Int J Cancer* 19(3): 291-297; 1977.

Selective and nonselective cytotoxicities were differentiated by applying interaction analysis to the results of an array of tests using four effector cells (of human, mouse and rat origin) and several target cells (TC). The latter comprised human tumor lines, 4 lymphoblastoid lines, 4 mouse tumor and nontumor cell lines, and 4 rat sarcoma cell lines. Nonselective effects were estimated from the two-way analysis of variance, and selective effects were calculated as the difference between the estimated nonselective and the observed reaction. The apparently nonselective effects of natural cytotoxicity were found to be actually highly specific. Cross competition assay results indicated that nonselective cytotoxicity is composed of many specific reactions against different TC. Each effector suspension included natural effector cells (N cells) that recognized and reacted specifically with several different antigens on TC, resulting in overall nonselectivity. The specificity of the reaction for each TC was demonstrated by the selective inhibition of natural cytotoxicity when competitor cells sharing antigens with the TC were added. Sharing of common target antigens was demonstrated by selective cross-inhibition, which was then used to investigate antigen on TC. The similarities found between man and rodent cell indicate a homology for natural cell-mediated cytotoxic reactions. (17 refs.)

**77-2156 Inhibitory Effect of Allogeneic Spleen Cells on Tumor Growth (Meeting Abstract).** (Eng.) Auerbach, R. (Dept. Zoology, Univ. Wisconsin, Madison, WI 53706) Sidky, Y. A. *Fed Proc* 36(3): 1204; 1977. (no refs)

**77-2157 Response of Lysine Rich Histones from Spleen to Subcutaneous Tumor (Meeting Abstract)** (Eng.) Mashburn, L. T. (Res. Inst., Hosp. Joint Disease New York, NY 10035) *Fed Proc* 36(3): 784; 1977. (no refs)

**77-2158 Cytotoxic Responses Between H-2 Identical M Locus Differing Mice (Meeting Abstract)** (Eng.) Kastner, D. L. (Baylor Coll. Medicine, Houston, TX 77030) Rich, R. R. *Fed Proc* 36(3): 1195; 1977. (no refs.)

- 77-2159 **Cytotoxic Effector Lymphocytes Can Be Selectively Depleted with Anti-Fluorescein Columns (Meeting Abstract).** (Eng.) Singer, K. H. (Duke Univ. Medical Center, Durham, NC 27710) Amos, D. B.; Scott, D. W. *Fed Proc* 36(3): 1280; 1977. (no refs.)
- 77-2160 **IgM and IgG Induced Cell Mediated Cytotoxicity with Human Lymphocytes as Effector Cells (Meeting Abstract).** (Eng.) Fuson, E. W. (Dept. Surgery and Microbiology, Univ. Alabama in Birmingham, Birmingham, AL 35294) Lamon, E. W. *Fed Proc* 36(3): 1324; 1977. (no refs.)
- 77-2161 **Marrow Dependent (M) Cells are Effectors in the Mitogen Induced Cellular Cytotoxicity (MICC) Assay (Meeting Abstract).** (Eng.) Yonkosky, D. (Boston Univ. Sch. Medicine, Boston, MA 02118) Bennett, M.; Cathcart, E. *Fed Proc* 36(3): 1325; 1977. (no refs.)
- 77-2162 **Tumor Inhibition by Effector Cells Either In Vitro or Cultured from Progressing Sarcomas (Meeting Abstract).** (Eng.) Bercezi, I. (Dept. Immunology, Univ. Manitoba, Winnipeg, Canada, R3E OW3) Schon, A. H. *Fed Proc* 36(3): 1306; 1977. (no refs.)
- 77-2163 **Inhibition of ADCC by Splenic Effector Cells from Rats Pretreated with Antigen and Antibody (Meeting Abstract).** (Eng.) Finnegan, A. (Surgical Res. Lab., Harvard Medical Sch., Boston, MA 02115) McConary, J. N. *Fed Proc* 36(3): 1280; 1977. (no refs.)
- 77-2164 **A Subclass of Human T Cells is Activated in MLC to Effect ADCC (Meeting Abstract).** (Eng.) Evans, R. L. (Sidney Farber Cancer Inst., Boston, MA 02115) Chess, L. *Fed Proc* 36(3): 1211; 1977. (no refs.)
- 77-2165 **A Comparison of Murine and Human Lymphoid Cell Populations as Sources of Effector Cells in Antibody-Dependent Cellular Cytotoxicity (ADCC) (Meeting Abstract).** (Eng.) Berger, A. E. (Duke Univ. Medical Center, Durham, NC 27710) *Fed Proc* 36(3): 1280; 1977. (no refs.)
- 77-2166 **Allogeneically Sensitized Cytolytic T Lymphocytes Specifically Lyse TNP Derivatized Targets H-2 Identical to the Effectors (Meeting Abstract).** (Eng.) Lemonnier, F. (Harvard Medical Sch., Boston, MA 02115) Burakoff, S.; Germain, R.; Benacerraf, B. *Fed Proc* 36(3): 1202; 1977. (no refs.)
- 77-2167 **Cellular Cytotoxicity Due to Cell Fragments After Induction by Calcium Ionophore A23187 (Meeting Abstract).** (Eng.) MacDermott, R. P. (Walter Reed Army Inst. Res., Washington, DC 20012) Nash, G. S.; Saint, J. G.; Bertovich, M. J. *Fed Proc* 36(3): 1281; 1977. (no refs.)
- 77-2168 **Induction of Cytotoxic T Cells After Treatment of Primed Splenocytes with Neuraminidase and Galactose Oxidase (Meeting Abstract).** (Eng.) Koppers, R. C. (The Johns Hopkins Univ., Sch. Medicine, Baltimore, MD 21239) Henney, C. S. *Fed Proc* 36(3): 1281; 1977. (no refs.)
- 77-2169 **Murine Sarcoma Virus (MSV) Induced T Cell Cytotoxicity: Lack of H-2 Restriction at the Sensitization or Effector Phase (Meeting Abstract).** (Eng.) Holden, H. T. (Lab. Immunodiagnosis, NCI, NIH, Bethesda, MD 20014) *Fed Proc* 36(3): 1202; 1977. (no refs.)
- 77-2170 **Cytotoxic Response of SJL Mice to Syngeneic Reticulum Cell Sarcomas (Meeting Abstract).** (Eng.) Roman, J. M. (Dept. Microbiology and Immunology, UCLA, Los Angeles, CA 90024) Bonavida, B. *Fed Proc* 36(3): 1306; 1977. (no refs.)
- 77-2171 **Different Cytotoxic Activities of Lymphoid Cell Preparations from Tumor-Immune VS. Tumor-Bearing Mice (Meeting Abstract).** (Eng.) Tokuda, S. (Univ. New Mexico, Sch. Medicine, Albuquerque, NM 87131) Vincent, J. *Fed Proc* 36(3): 1306; 1977. (no refs.)
- 77-2172 **Establishment and Characterization of the R3327 Prostatic Adenocarcinoma In Vitro (Meeting Abstract).** (Eng.) Reynolds, C. W. (Univ. Iowa and V.A. Hosp., Iowa City, IA 52240) Rasmussen, G. T.; Lubaroff, D. M.; Porter, J. R. *Fed Proc* 36(3): 1292; 1977. (no refs.)
- 77-2173 **Immunogenicity of the Dunning R3327 Prostate Adenocarcinoma in the Fisher-Copenhagen F<sub>1</sub>**



**Hybrid Rat (Meeting Abstract).** (Eng.) McKinney, E. C. (Dept. Microbiology, Univ. Miami, FL 33152) Fletcher, M. A.; Block, N. L.; Claflin, A. J. *Fed Proc* 36(3): 1292; 1977. (no refs.)

**77-2174 Tumour-Associated Immunity in Prostatic Cancer (Meeting Abstract).** (Eng.) Ablin, R. J. (Cook County Hosp., Chicago, IL 60612) Guinan, P. D.; Bhatti, R. A. *Fed Proc* 36(3): 1256; 1977. (no refs.)

**77-2175 Characterization of the CIQ-Reactive Material in Cancer Patients' Sera (Meeting Abstract).** (Eng.) Reisberg, M. A. (V.A. Hosp. and Baylor Coll. Medicine, Houston, TX 77211) Rossen, R. D. *Fed Proc* 36(3): 1257; 1977. (no refs.)

**77-2176 Studies on Immunity to Mouse Sarcoma Using the Tumor-Cell Neutralization Test.** (Eng.) Thomas, H. C. (Div. Tumor Immunology, Fred Hutchinson Cancer Res. Center, 1124 Columbia St., Seattle, WA 98104) Hellstrom, K. E.; Kall, M. A.; Hellstrom, I.; van Belle, G. *Trans Am Acad Ophthalmol Otolaryngol* 84(2): 291-301; 1977.

The results of tumor-cell neutralization (Winn) tests were analyzed with respect to tumor incidence, time to palpable tumor (latency period), and growth rate (time to reach a specified size less the time to palpable tumor). The concept of relative risk and the Z statistic were used to describe the latter two variables. The analysis suggested that either latency period or tumor growth rate can be affected as a result of admixing lymph node cells with tumor cells prior to injection into mice. Experiments were then performed to determine if in vitro-sensitized lymphocytes would specifically inhibit the outgrowth of BALB/c 1315 and 1321 sarcoma cells in Winn tests. Lymphocytes from 1315- and 1321-immune animals significantly neutralized 1315 tumor cells. The 1315- and 1321-sensitized lymphocytes did not inhibit or stimulate tumor outgrowth, but they facilitated the growth rate. The importance of using latency period and growth rate as measures of cellular immunity is discussed. (20 refs.)

**77-2177 A Nude Mouse Tumor Neutralization Test Applied to Patients Suffering from Skin Melanoma. A Methodological Study.** (Eng.) Kristensen, E. (Fibiger Lab., Ndr. Frihavsgade 70, DK-2100 Copenhagen O, Denmark) *Proc Soc Exp Biol Med* 155(1): 27-30; 1977.

A nude mouse tumor neutralization test (TNT) was used to evaluate cell-mediated immunity in seven patients suffering from localized skin melanoma (Stages I and II). The target

cells were melanoma cells derived from an established in vitro cell line known to be tumorigenic in nude mice but without a tendency to invasive growth or the formation of distant metastases. For each of the seven patients, 30 mice were divided into three groups. Group 1 mice were inoculated with  $3 \times 10^6$  control lymphocytes (from 7 healthy donors) plus  $3 \times 10^5$  melanoma cells in a 0.1-ml volume of phosphate buffered saline. Group 2 animals received patient lymphocytes plus melanoma cells in the same amounts. Group 3 animals were inoculated with melanoma cells alone. There was a reduced number of takes in Group 2 mice (35 tumors compared to Groups 1 and 3 (47 and 46 tumors). The patient lymphocytes (Group 2) also increased the latency period significantly between weeks 2-5, but tumor growth was not affected. These findings support the assumption that lymphocytes from melanoma patients are sensitized against group-specific antigen. (28 refs.)

**77-2178 Inhibition of In Vitro Lymphocyte Function by Cyst and Ascitic Fluids from Ovarian Cancer Patients (Meeting Abstract).** (Eng.) Hess, A. (Duke Univ. Medical Center, Durham, NC 27710) Gall, S. *Fed Proc* 36(3): 1218; 1977. (no refs.)

**77-2179 Immunosuppressive Activity and Tissue Polypeptide Antigen (TPA) Content of Human Ascitic Fluid (Meeting Abstract).** (Eng.) Badger, A. M. (Boston Univ. Sch. Medicine, Boston, MA 02118) Buchler, R. A.; Cooperband, S. R. *Fed Proc* 36(3): 1319; 1977. (no refs.)

**77-2180 Depression of Anti-T Antibodies in Patients with Gastrointestinal Carcinoma (Meeting Abstract).** (Eng.) Desai, P. R. (Dept. Immunochimical Research, Evanston Hosp., Evanston, IL 60201) Springer, G. I.; McNeil, C. *Fed Proc* 36(3): 1254; 1977. (no refs.)

**77-2181 Depressed In Vitro Peripheral Blood Lymphocyte Response to Mitogens in Cancer Patients: The Role of Suppressor Cells.** (Eng.) Zembala, M. (Div. Microbiology and Immunology, Inst. Paediatrics, Wroclaw 265, 30-663 Cracow, Poland) Mytar, B.; Popiela, J.; Asherson, G. L. *Int J Cancer* 19(5): 605-613; 1977.

Mitogen-induced stimulation of protein synthesis as measured by  $^3\text{H}$ -leucine incorporation showed that the response of peripheral blood lymphocytes was depressed in patients with advanced malignancy. The lymphocytes of 15 cancer patients and one patient with Hodgkin's disease inhibited the reactivity of normal lymphocytes in cocultures. In contrast, lymphocytes from healthy subjects, patients with chronic lymphocytic leukemia, lymphosarcoma or multiple myeloma

caused no suppression. Purified T cells from patients with carcinoma responded to mitogens while unseparated lymphocytes failed to respond. There was no evidence for B-cell-mediated suppression. The inhibitory activity seemed to be due to adherent cells, presumably monocytes; however, in two cases inhibition was caused by isolated T cells of the patients and not by adherent cells. It is suggested that one mechanism for the depression of cell-mediated immunity seen in patients with advanced cancer may be the nonspecific suppression of certain T-cell functions by circulating monocytes. (40 refs.)

77-2182 **Plasma Inhibitor in T-Cell Leukemia (Meeting Abstract).** (Eng.) Mukhopadhyay, N. (Baylor Coll. Medicine, Houston, TX 77030) Shepherd, D. A.; Fernbach, D. J. *Fed Proc* 36(3): 1319; 1977. (no refs.)

77-2183 **Role of Pyridoxal Phosphate (PLP) Phosphatase Activity in Regulation of Plasma PLP Levels in Cancer Patients (Meeting Abstract).** (Eng.) Potera, C. (Dept. Human Oncology, Univ. Wisconsin Center Health Sciences, Madison, WI 53706) Brown, R. R.; Rose, D. P. *Fed Proc* 36(3): 1137; 1977. (no refs.)

77-2184 **Activity and Intracellular Localization of Lysosomal Acid Phosphatase in Lymphocytes from Patients with Hodgkin's Disease, Plasma Cell Myeloma and Primary Polycythemia.** (Eng.) Lisiewicz, J. (Hematological Clinic, Inst. Internal Medicine, Acad. Medicine, Cracow, Poland) Astaldi, G. *Tumori* 62(6): 651-657; 1976.

The activity and localization of lysosomal acid phosphatase (AP) in peripheral blood lymphocytes were studied cytochemically. The investigation comprised 20 healthy subjects (10 men, 10 women aged 20-30 yr) and 30 patients with malignancies (15 men, 15 women aged 40-56 yr), 10 with Hodgkin's disease, 10 with plasma cell myeloma, and 10 with primary polycythemia. In patients with Hodgkin's disease, the total lymphocyte count and the absolute count of AP-positive lymphocytes were lower than those in normal adults. Lymphocytes with a granular reaction containing one and two AP-positive granules were less numerous in the patients. A characteristic feature was the absence of lymphocytes with three and four AP-positive granules. The total count of AP-positive lymphocytes with a mixed reaction type and with a single AP-positive granule was lower in the patients. An important feature was the absence of cells with this enzymatic reaction type having two to five or more AP-positive lysosomal granules. Analogous changes in lymphocytes from patients with primary polycythemia and those with plasma cell myeloma were much less significant. The results are important for assessment on a cellular basis of the lowered immunity of patients with myeloproliferative and lymphoprolifera-

tive disorders, with special regard to the use of AP as a T-cell marker. (19 refs.)

77-2185 **N-Acetyl-beta-glucosaminidase of Peripheral Blood Lymphocytes in Patients with Cancer of the Larynx.** (Eng.) Gierak, T. (Clinic Laryngology, Inst. Nervous System and Sensory Perception Organ Diseases, Silesian Medical Acad., Katowice, Poland) Astaldi, G.; Lisiewicz, J.; Pilch, J. *Tumori* 62(6): 645-650; 1976.

The N-acetyl- $\beta$ -glucosaminidase (GS) activity in the peripheral blood lymphocytes of 20 men (aged 35-55 yr) with cancer of the larynx was assessed cytochemically. Av values of the absolute lymphocyte count in these patients and in healthy subjects (20 men, aged 20-30 yr) did not differ significantly. In contrast, the av absolute count of GS-negative lymphocytes was significantly lower in the cancer patients than in the controls. The GS-positive lymphocytes, which had a granular-type reaction and one to two enzyme-positive granules, were less numerous in the patients than in the controls. The numbers of lymphocytes with three to five GS-positive granules did not differ between the patients and control subjects. The total count of GS-positive lymphocytes with a granular-type reaction was nearly twice as high in the controls as in the patients. GS-positive lymphocytes with a granular-diffuse-type reaction and 1, 2, 3, and 5 or more enzyme-positive granules were more numerous in the patients. The total count of lymphocytes with this reaction was strikingly higher in the patients than in the controls (mean values were 305.0 and 45.9 cells/mm<sup>3</sup>, respectively). GS-positive lymphocytes with a diffuse type of cytochemical reaction were significantly more numerous in the patients. The changes may be related to the immune reaction of specific tumor antigens. (19 refs.)

77-2186 **Serum Ribonuclease in Patients with Lung Carcinoma.** (Eng.) Marabella, P. C. (Dept. Thoracic Surgery, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY) Tritsch, G. L.; Moore, R. H.; Takita, H. *J Surg Oncol* 8(6): 501-505; 1976.

Serum ribonuclease (RNase)-level determinations were performed on 51 patients with untreated primary bronchogenic carcinoma, 24 with localized and 27 with disseminated disease. Histologically, there were 9 squamous cell, 9 oat cell, 8 large cell, 4 anaplastic, and 21 adenocarcinomas. Five employees and three referred patients with no evidence of disease were used as controls. The mean RNase level (in optical density units/ml serum) in all lung cancer patients was 28.0, compared to 21.5 for controls. The 24 localized cases had a mean RNase level of 25.4, compared to 30.3 for the 27 disseminated cases. In addition, 8/12 localized cases tested by WBC migration inhibition had positive inhibition. The mean RNase level was 24.4. Nine of the 10 disseminated cases had negative inhibition, with a mean RNase of 30.5. The adeno-



carcinomas had a mean RNase of 27.3, the squamous cell carcinomas 28.1, the oat cell carcinomas 30.4, the large cell carcinomas 28.7, and the anaplastic carcinomas 24.2. WBC migration inhibition results indicate that there is an impairment of cellular immunity in cases with more elevated RNase levels. RNase may be implicated in a blocking phenomenon associated with neoplastic disease. (8 refs.)

**77-2187 Six Myeloma Protein Products from a Single Patient (Meeting Abstract).** (Eng.) Jemmerson, R. (Northwestern Univ., Evanston, IL 60201) Kaplan, B.; Urbanski, G. J. *Fed Proc* 36(3): 1197; 1977. (no refs.)

**77-2188 Isolation and Characterization of the Zinc Glycinate Marker (ZGM) of Colon Cancer (Meeting Abstract).** (Eng.) Oh, S. K. (Mallory Gastrointestinal Res. Lab., Sears Surgical Lab., Boston City Hosp., Boston, MA 02118) Saravis, C. A.; Doos, W.; Zamcheck, N. *Fed Proc* 36(3): 1326; 1977. (no refs.)

**77-2189 A Novel Glycoprotein in the Urine of a Patient with Carcinoma of Colon (Meeting Abstract).** (Eng.) Chawla, R. K. (Emory Univ., Atlanta, GA 30322) Heymsfield, S. B.; Wadsworth, A. D.; Shoji, M.; Rudman, D. *Fed Proc* 36(3): 1326; 1977. (no refs.)

**77-2190 Immunosuppressive Proteins in the Serum of Breast Cancer Patients (Meeting Abstract).** (Eng.) Ninnemann, J. L. (Univ. California, San Diego, Sch. Medicine, La Jolla, CA 92037) Fisher, J. C. *Fed Proc* 36(3): 1270; 1977. (no refs.)

**77-2191 Immunological Studies on Progress of Diseases from Viral Hepatic to Hepatomas (Meeting Abstract).** (Eng.) Kuroki, T. (Third Dept. Internal Medicine Osaka, City Univ. Medical Sch., Osaka, Japan) Kawai, K. *Gastroenterol Jpn* 12(1): 83-84; 1977. (no refs.)

**77-2192 Secondary Immunoproliferative Disease in Hamsters Bearing Human Tumors: Resemblance to Human Immunoblastic Lymphadenopathy (Meeting Abstract).** (Eng.) Richman, A. V. (Dept. Pathology, Coll. Medicine, Univ. Florida, Gainesville, FL 32610) *Fed Proc* 36(3): 1053; 1977. (no refs.)

**77-2193 Effects of Bursectomy on the Incidence of Rous Sarcomas in Genetically Resistant Chickens**

**(Meeting Abstract).** (Eng.) Watanabe, D. H. (New York Medical Coll., Valhalla, NY 10595) Schierman, L. W.; McBride, R. A. *Fed Proc* 36(3): 1222; 1977. (no refs.)

**77-2194 Effect of Splenectomy on Host Resistance to Cancer (Meeting Abstract).** (Eng.) Chang, R. W. (Dept. Pathology, Royal Coll. Surgeons England, London, England) Turk, J. L. *Br J Surg* 64(4): 291; 1977. (no refs.)

**77-2195 Oncogenesis in Congenitally Asplenic Mice (Eng.) Fletcher, M. P.; Ikeda, R. M.; Gershwin M. E. In: *Immuno-aspects of the Spleen. Proceedings of a Conference on Immunodynamics, Cleveland, Ohio, May 19 and 20, 1976.* Battisto, J. R.; Streilein, J. W., eds. (New York: North-Holland Publishing Co.): pp. 377-389; 1976.**

The incidence of spontaneous tumors was investigated in a colony of 321 congenitally asplenic and 381 normal littermate control mice, and their susceptibility to both Moloney murine sarcoma virus (M-MSV)- and dimethylbenzanthracene (DMBA)-induced tumors was compared. There were no significant neoplasia during the 1-yr observation period. There was an increased incidence of hamartomas in the asplenic colony (5%) compared to the controls (0.1%). The hamartomas were generally found in the pelvis and abdomen, particularly in the region of the urogenital tract. There was no relationship between the occurrence of hamartomas and either polycystic kidneys or major hind-limb abnormalities. The prevalence of polycystic kidneys was 25% in the asplenic colony and < 0.1% in the controls. Following an injection of M-MSV, the latent period for tumor development was the same in both groups (8 days), but the tumor was larger and the regression time longer in the asplenic animals than in the controls. Asplenic mice were less susceptible than control to papilloma induction by skin painting with DMBA. (3 refs.)

**77-2196 Tumor Enhancement in Absence of Spleen (Eng.) Pasqualini, C. D.; Colmerauer, M. E. In: *Immuno-aspects of the Spleen. Proceedings of a Conference on Immunodynamics, Cleveland, Ohio, May 19 and 20, 1976.* Battisto, J. R.; Streilein, J. W., eds. (New York: North Holland Publishing Co.): pp. 415-419; 1976.**

In two different tumor-host combinations, the AKR lymphoma in BALB mice and sarcoma 180 in Swiss mice, immunological tumor enhancement was demonstrated in splenectomized animals. The AKR lymphoma donor tumor L15, did not grow in BALB mice when it was implanted s.c. or i.p. The mere implantation of a glass cylinder also did not lead to tumor development. However, it constituted a privileged site because the injection of L15 within 2 days later led to the development of an allogeneic tumor (lymphoma P) i

66% of the animals, killing them in 37 days. Splenectomy led to a decreased lymphoma P incidence (41%). Tumor antigen pretreatment increased the incidence of lymphoma P to 92%, a result that was not altered by splenectomy. An sc challenge of Swiss mice with sarcoma 180 (S180) led to a high rate of spontaneous rejection, with only 23% of the animals dying of tumor. Injection of S180 within the glass cylinder increased tumor incidence to 68%. Similar tumor incidence was obtained by pretreating the host with acellular S180 extracts 10 days before an sc challenge with viable tumor cells. When both procedures were combined, 95% of the animals died with large tumors. Splenectomy decreased the incidence of S180 injected either within the glass cylinder or sc to 30% and 12%, respectively. Whenever the animals received tumor antigen pretreatment, however, splenectomy had no effect. The spleen is not essential for active immunological enhancement, and soluble tumor antigen pretreatment can override the regression effect of splenectomy. (17 refs.)

77-2197 **Immunologic Effects of Endogenous Virus-Producing Tumors in Thymectomized or Splenectomized BALB/c Mice (Meeting Abstract).** (Eng.) Barker, A. D. (Battelle, Columbus Labs., Columbus, OH 43201) Dennis, A. J.; Moore, V. S. *Fed Proc* 36(3): 1291; 1977. (no refs.)

77-2198 **Cellular Immunotherapy of Post-Thymectomy Spontaneous Lymphomas (Meeting Abstract).** (Eng.) Cornelius, E. A. (Yale Univ. Sch. Medicine, New Haven, CT 06510) *Fed Proc* 36(3): 1290; 1977. (no refs.)

77-2199 **Thymus Mediated Suppression of Resistance to Tumor Growth (Meeting Abstract).** (Eng.) Samuel, E. S. (Dept. Surgery, Univ. Toronto, Toronto, Ontario, Canada MSG 1A8) Mossal, N.; Falk, J. A.; Falk, P. E. *Fed Proc* 36(3): 1205; 1977. (no refs.)

77-2200 **Depression of Host Immune Response by a Tumor Associated Virus (Meeting Abstract).** (Eng.) Campbell, D. A. (Univ. Michigan and NIH, Bethesda, MD) Niederhuber, J. E.; Manders, E. D.; Herberman, R. B. *Fed Proc* 36(3): 1204; 1977. (no refs.)

77-2201 **Viral Suppression of Neutrophil Chemotaxis in Man (Meeting Abstract).** (Eng.) Park, B. H. (State Univ. New York at Buffalo and Children's Hosp., Buffalo, NY 14222) Chiba, Y.; Ramirez, R. I.; Ogra, P. L. *Fed Proc* 36(3): 1189; 1977. (no refs.)

77-2202 **Suppressed Rosette Cell Formation Following Oncornavirus Induced Brain Tumors or Infections In Nonhuman Primates (Meeting Abstract).** (Eng.) Johnson, L. (Rush and Univ. Illinois Medical Centers, Chicago, IL 60612) Wolfe, L. G.; Deinhardt, F. *Fed Proc* 36(3): 1291; 1977. (no refs.)

77-2203 **Ability of Serum Factors to Enhance Growth of an SV40 Induced Tumor in Hamsters (Meeting Abstract).** (Eng.) Hunter, R. (Dept. Pathology and the Franklin McLean Memorial Res. Inst., Univ. Chicago, Chicago, IL 60637) Strickland, F. *Fed Proc* 36(3): 1204; 1977. (no refs.)

77-2204 **Effects of Immunoactive Substances on Murine Virus Leukemogenesis and Immunosuppression.** (Eng.) Ceglowski, W. S.; Tripodi, D.; Strauss, R. R.; Friedman, H. In: *Modulation of Host Immune Resistance in the Prevention or Treatment of Induced Neoplasias*. NCI and John E. Fogarty International Center for Advanced Study in the Health Sciences. (Bethesda, Maryland): pp. 287-293; 1974.

The ability of immunoactive substances to alter viral leukemogenesis and leukemia virus-induced immune depression was investigated. Friend leukemia virus (FLV) was used as the tumorigenic agent. Mice infected with FLV demonstrated a significant impairment in their ability to respond to an antigen such as sheep RBC, as measured by decreased numbers of antibody plaque-forming cells and by decreased titers of hemagglutinating antibody. Levamisole restored immune competence in mice when administered soon after infection with FLV. When thioglycollate medium was given to mice 2 days prior to FLV, it suppressed leukemogenesis significantly. When administered 1 day before virus, carbon particles suppressed leukemogenesis moderately, presumably because of its effect on macrophages. The potentiation of leukemogenesis occurred when BCG, complete Freund's adjuvant, and incomplete Freund's adjuvant were administered before virus. The effects seem to be due to a complex interaction between the cells of the immune system and leukemia viruses. (22 refs.)

77-2205 **Nonspecific Immunosuppression and Expression of Avian Myeloblastosis Virus (BAI Strain A).** (Eng.) Smida, J. (Cancer Res. Inst., Slovak Acad. Sciences, 880 32 Bratislava, Czechoslovakia) Smidova, V. *Neoplasma* 23(6): 589-594; 1976.

The nonspecific expression and immunosuppression of avian myeloblastosis virus (BAI strain A) were investigated. Cyclophosphamide (CP; im or ip) was well-tolerated by 7- to 15-day-old White Leghorn chickens (phenotype C/O) when the



total dose of the drug was  $\leq 25$  mg. Chickens were challenged with virus on days 1 or 2 after the CP administration was completed. Virus was also administered to control, untreated, birds. There was a strong age-related resistance to the myeloblastosis: untreated control birds at 2 wk of age and older were fully resistant to the disease. However, the development of myeloblastosis in treated and untreated 1-day-old chickens was very similar. The appearance of the first myeloblasts in blood smears was observed approx 8 days after virus injection. The myeloblastosis proceeded rapidly, and the terminal stage of the disease was reached approx 2 wk after challenge with virus. Some chickens from the treated group, with myeloblastosis, died suddenly; they showed massive infiltration of the liver and spleen with myeloblasts. The plasmas of all other animals with myeloblastosis demonstrated high levels of adenosine triphosphatase activity. During 60 days of observation, no neoplastic lesions were found in the chickens except for acute myeloblastic leukemia. The high incidence of myeloblastosis in 1-day-old chicks after transplantation of myeloblasts was almost identical to the response of the corresponding group of birds receiving virus. In comparison to the drug-treated group injected at the same age with virus, the incidence of myeloblastosis was lower in the group receiving myeloblasts. The level of ATP in diseased birds showed 2.8-3.9  $\mu$ moles Pi released/min/ml of viremic plasma. Because of the greater volume of the viremic plasmas obtained from older animals, the yield of virus from a single bird was five to seven times higher than that usually found in chicks injected with virus at the first 3 days of age. The results indicate that nonspecific immunosuppression by CP treatment significantly affects virus expression in age-resistant chickens. (11 refs.)

**77-2206 Immunosuppressive Cells in Moloney Leukemia Spleen: Are They Leukemic Cells or Normal Cells (Meeting Abstract)?** (Eng.) Grinwich, K. D. (Dept. Micro., Harvard Sch. Public Health, Boston, MA 02115) Stiller, R. A.; Cerny, J. *Fed Proc* 36(3): 1269; 1977. (no refs.)

**77-2207 Immunosuppression Induced by the Murine Lymphoma, FBL-3 (Meeting Abstract).** (Eng.) Specter, S. (Albert Einstein Medical Center, Phila., PA 19141) Cimprich, R.; Farber, P.; Friedman, H. *Fed Proc* 36(3): 1274; 1977. (no refs.)

**77-2208 Subversion of Host Defense Mechanisms by Malignant Tumors: An Established Tumor as a Privileged Site for Bacterial Growth.** (Eng.) Spitalny, G. L. (Trudeau Inst., Saranac Lake, NY 12983) North, R. J. *J Exp Med* 145(5): 1264-1277; 1977.

The hypothesis that an established primary tumor presents an environment that is not only antagonistic to the expression of antitumor immunity but is also antagonistic to the expression of host defenses in general was assessed. The SA1 spindle cell sarcoma syngeneic in A/J mice, the Meth A fibrosarcoma syngeneic in BALB/c mice, and the P815 mastocytoma syngeneic in DBA/2 mice were studied. Mice carrying any one of the tumors in their right hind foot pad were as capable as control mice of eliminating *Listeria monocytogenes* from their contralateral tumor-free foot pad, lymph nodes, and livers. However, they were incapable of eliminating an inoculum of the organism from the progressive tumor. A similarity exists between the capacity of a tumor-bearing animal to express T-cell-mediated concomitant antitumor immunity and its capacity to express T-cell-mediated antibacterial immunity, in that although both can be efficiently expressed systematically, neither can be fully expressed within the growing tumor. (26 refs.)

**77-2209 Immunosuppression of the 1° and 2° Immune Response by a  $\gamma$ M Plasmacytoma (TEPC-183) (Meeting Abstract).** (Eng.) Fenton, M. R. (Temple Univ., Philadelphia, PA 19140) Havas, H. F.; Schiffman, G.; Berner, S.; Goodis, A. *Fed Proc* 36(3): 1274; 1977. (no refs.)

**77-2210 Effect of Immunosuppression on Spontaneous Mammary Tumorigenesis in Mice.** (Eng.) Nagasawa, H. (Pharmacology Div., Natl. Cancer Center Res. Inst., Tsukiji 5-1-1, Chuo-ku, Tokyo 104, Japan) *IRCS Med Sci: Cancer* 5(5): 219; 1977.

The effect of immunosuppression alone or in combination with pituitary grafting on spontaneous mammary tumorigenesis is reported. Sixty-day-old SLN female mice were divided into four groups: Group I served as a control and received no treatment, Group II was given ip injections of Imuran, Group III was grafted with two isologous pituitaries each under the kidney capsules, and Group IV received both the pituitary grafts and the Imuran injections. Mice in Groups I and II failed to show palpable mammary tumors by 10 mo of age, but all the mice in group IV had tumors. The incidence of mammary tumors increased with age in Groups III and IV. Group IV, which had the additional treatment with Imuran, showed the highest incidence of mammary tumors. The results suggest that immunosuppression accelerates the carcinogenic effects of both known and unknown factors, but has little action by itself. (5 refs.)

**77-2211 Cell Interaction in the Suppression of Primed Immune Responses In Vitro (Meeting Abstract).** (Eng.) Oalkins, C. P. (Sloan-Kettering Inst., New York, NY 10021) Stanton, T.; Stutman, O. *Fed Proc* 36(3): 1184; 1977. (no refs.)

- 77-2212 **An Analysis of the Immunosuppressive Activity of Human and Mouse Alpha-Fetoprotein (Meeting Abstract).** (Eng.) Murgita, R. A. (Dept. Immunology, Univ. Uppsala, Sweden) Andersson, L. C.; Engvall, E.; Ruoshtti, E.; Wigzell, H. *Fed Proc* 36(3): 1318; 1977. (no refs.)
- 77-2213 **Conditions Altering the Immunosuppressive Activity of Human Alpha-Fetoprotein (Meeting Abstract).** (Eng.) Gocken, N. E. (Univ. Iowa and the V.A. Hosp., Iowa City, IA 52240) Benno, R. H.; Thompson, J. S. *Fed Proc* 36(3): 1319; 1977. (no refs.)
- 77-2214 **The Effect of Alpha Fetoprotein (AFP) on Immune Phenomena in Rats and Mice (Meeting Abstract).** (Eng.) Sheppard, H. W. (Univ. Calif. San Diego, Jolla, CA) Sell, S.; Trefts, P.; Poler, S. M.; Bahu, R. *Fed Proc* 36(3): 1318; 1977. (no refs.)
- 77-2215 **Trypan Blue Prevents Immunologically-Mediated Tumor Rejection (Meeting Abstract).** (Eng.) Kreider, J. W. (Dept. Pathology, Hershey Medical Center, Hershey, PA 17033) Bartlett, G. L. *Fed Proc* 36(3): 123; 1977. (no refs.)
- 77-2216 **Inhibition of In Vitro Parameters of Immunity by Synthetic Polyamines (Meeting Abstract).** (Eng.) Byrd, W. (Salk Inst., San Diego, CA 92112) Jacobs, M. *Fed Proc* 36(3): 1230; 1977. (no refs.)
- 77-2217 **Immunosuppression and Heightened Cyclic AMP Induced by Tobacco Smoke Products (Meeting Abstract).** (Eng.) Meyers, S. R. (Dept. Microbiology, Univ. Louisville Sch. Medicine, Louisville, KY 40201) Elzer, G. T.; Jacob, C. V.; Wallace, J. H. *Fed Proc* 36(3): 130; 1977. (no refs.)
- 77-2218 **Tumor Regression Pattern and Host Cell Infiltrate in Fibrosarcoma Growing in Normal and Immunosuppressed C3H Mice (Meeting Abstract).** (Eng.) Andiondo, O. (Edwin L. Steele Lab. Radiation Biology, Dept. Radiation Medicine, Massachusetts General Hosp., Boston, MA) Suit, H. D.; Bahn, A. K. *Int J Radiat Oncol Biol Phys* 1(Suppl 1): 46; 1976. (no refs.)
- 77-2219 **Lack of H-2 Restriction in Rejection of Homografts (Meeting Abstract).** (Eng.) Murasko, D. M. (The Jackson Lab., Bar Harbor, ME 04609) *Fed Proc* 36(3): 1203; 1977. (no refs.)
- 77-2220 **Non-Correlation Between Transplantation Behavior and H-2 Antigen Expression of Congenic Methylcholanthrene-Induced Fibrosarcomas (Meeting Abstract).** (Eng.) Chauvenet, P. H. (Tumor Biology Unit, Univ. Florida, Gainesville, FL 32610) Smith, R. T. *Fed Proc* 36(3): 1203; 1977. (no refs.)
- 77-2221 **C'3 Participation in the Rejection of Some Experimental Tumors.** (Eng.) Bellelli, L. (Regina Elena Inst. Cancer Res., Lab. Physiopathology, Viale Regina Elena 291, I-00161 Rome, Italy) Sezzi, M. L. *Oncology* 33(5/6): 215-218; 1976.
- The possibility that C'3 participates in tumor rejection was assessed in DBA/2 mice previously immunized against L1210 leukemia and in Swiss mice previously immunized against Ehrlich's adenocarcinoma. In both strains, rosette formation was inhibited by cobra venom factor (CoF) treatment in immunized and nonimmunized mice. Only mice previously immunized and not treated with CoF rejected the challenge of tumor cells, and they were alive some months later, with no signs of neoplasia. Immunized mice treated with CoF did not reject the challenge, and a neoplasia began to appear 1 wk later. The av survival time of immunized animals treated with CoF was approx twice that of nonimmunized mice treated or not treated with CoF. Microscopic observation of the peritoneal washing fluids demonstrated that in nonimmunized animals, the number of cancer cells increased progressively until they formed the majority of the cell population. In C'3-depleted immunized mice, immunoglobulin M was present on tumor cells, macrophages, and lymphocytes, as in immunized controls. However, no contact between the cells and no macrophage phagocytosis was noted, and the number of tumor cells increased progressively. C'3 is concluded to be involved in the rejection of the experimental tumors. (16 refs.)
- 77-2222 **Involvement of Lymphocyte Mediators in the Rejection of a Murine Tumor Allograft.** (Eng.) Harrington, J. T. (Dept. Medicine, Veterans Admin. Hosp., Univ. Texas Health Science Center, San Antonio, TX 78284) *Cell Immunol* 30(2): 261-271; 1977.
- Macrophage migration inhibition by peritoneal WBC was studied in BALB/c mice bearing ip allogeneic EL-4 lymphomas to explore the role of this immune effector function in allograft rejection. BALB/c mice were inoculated ip with  $3 \times 10^7$  EL-4 tumor cells, and peritoneal WBC were harvest-



ed 8-10 days after inoculation. The WBC population was fractionated to separate adherent macrophages from nonadherent cells, including lymphocytes and lymphoma cells that were not morphologically distinguishable. The nonadherent population inhibited the migration of nonimmune murine macrophages, as demonstrated by direct and indirect migration assays using the agarose droplet method. This host response also contained large numbers of adherent macrophages, which had been shown previously to be cytotoxic to EL-4 target cells. These findings provide evidence for lymphokine activity in allograft rejection and suggest that lymphocyte mediators may attract and activate the cytotoxic macrophages observed in this response. (35 refs.)

**77-2223 Factors Regulating the In Vivo Graft-Versus-Host Reaction as Assayed by Lymphocyte-Induced Angiogenesis (Meeting Abstract).** (Eng.) Sidky, Y. A. (Dept. Zoology, Univ. Wisconsin, Madison, WI 53706) Auerbach, R. *Fed Proc* 36(3): 1062; 1977. (no refs.)

**77-2224 Suppression of Graft-Versus-Host Reactions (GVH) in Mice by Vesicular Stomatitis Virus (Meeting Abstract).** (Eng.) Romano, T. J. (New York Univ. Sch. Medicine, New York, NY 10016) Umetsu, D. T.; Bloom, B. B.; Thorbecke, G. J.; Hochwald, G. M. *Fed Proc* 36(3): 1192; 1977. (no refs.)

**77-2225 Production of Specific Macrophage Activating Factor by Lymphocytes from Tumor-bearing Mice.** (Eng.) Kripke, M. L. (Basic Res. Program, NCI Frederick Cancer Res. Center, Frederick, MD 21701) Budmen, M. B.; Fidler, I. J. *Cell Immunol* 30(2): 341-352; 1977.

The production of macrophage-activating factor (MAF) in tumor-bearing mice was investigated using a recent UV-induced fibrosarcoma (UV-112). Lymphocytes from C57BL mice bearing the UV-112 tumor produced MAF when they were cultured with UV-112 cells. This MAF rendered normal C57BL macrophages cytotoxic to UV-112 cells in vitro. Both MAF production and lymphocyte-mediated growth inhibition were present during the early stages of tumor growth. They disappeared when the tumors reached an advanced stage, implying that the failure of lymphocytes from mice bearing large tumors to produce MAF was not due to a lack of sensitization but rather to the presence of a large tumor mass. This eclipsed reactivity was specific for the growing tumor. Lymphocytes from mice bearing a large UV-112 tumor were still able to produce MAF in response to the B16 melanoma to which they had been preimmunized. The MAF released by C57BL lymphocytes in response to either B16 or UV-112 was specific, in that it rendered macrophages cytotoxic only against the tumor used for immunization. This raises the possibility that there may be more than one sub-

stance (specific and nonspecific) capable of activating macrophages to become cytotoxic. (33 refs.)

**77-2226 The Effect of Agents on Adherence and Phagocytosis in P388 D<sub>1</sub> Cells in Tissue Culture (Meeting Abstract).** (Eng.) Goodell, E. (Dept. Pharmacology, Medical Coll. Virginia, Virginia Commonwealth Univ., Richmond, VA 23298) Kessler, F.; Bilgin, S.; Carchman, R. A. *Fed Proc* 36(3): 1252; 1977. (no refs.)

**77-2227 Stimulation of Phagocytic Activity of Granulocytes by Supernatant Fluids from Human Lymphocyte Cultures (Meeting Abstract).** (Eng.) Klostergaard, J. (Roswell Park Memorial Inst., Buffalo, NY 14263) Klein, E.; Holtermann, O. A. *Fed Proc* 36(3): 1280; 1977. (no refs.)

**77-2228 Macrophage Function During Friend Virus-Induced Leukemia and Its Spontaneous Regression (Meeting Abstract).** (Eng.) Marcelletti, J. (Dept. Biology, Michigan Cancer Foundation, Detroit, MI 48201) Furmanski, P. *Fed Proc* 36(3): 1262; 1977. (no refs.)

**77-2229 Macrophage Shortage Within Tumor Diminishes Effectiveness of IgG1 Antibody-Mediated Suppression (Meeting Abstract).** (Eng.) Johnson, R. J. (Johns Hopkins Univ. Sch. Medicine, Baltimore, MD 21205) Economou, J. S. *Fed Proc* 36(3): 1256; 1977. (no refs.)

**77-2230 Alteration of Macrophage Function During Tumor Growth (Meeting Abstract).** (Eng.) Jones, T. L. (Michigan State Univ., East Lansing, MI 48824) *Fed Proc* 36(3): 1256; 1977. (no refs.)

**77-2231 The Requirement for H-2 Compatible Macrophages for the Generation of Migration Inhibition Factor by Immune T Cells in Response to Soluble Tumor Antigens (Meeting Abstract).** (Eng.) Landolfo, S. (NIH, Bethesda, MD 20014) Herberman, R. B. *Fed Proc* 36(3): 1202; 1977. (no refs.)

**77-2232 Localization of Ingested Particles in Macrophages of Gut-Associated Lymphoid Tissues (Meeting Abstract).** (Eng.) Joel, D. D. (Dept. Medicine, Brookhaven Natl. Lab., Upton, NY 11973) Chanana, A. D.;

ronkite, E. P.; Laissue, J. A.; LeFevre, M. E. *Fed Proc* 36(3): 201; 1977. (no refs.)

7-2233 **Enhancement of Lewis Lung Carcinoma in a Syngeneic Host by Spleen Cells of C57BL/6 Mice.** (Eng.) Gozes, Y. (Dept. Cell Biology, Weizmann Inst. of Science, Rehovot, Israel) Trainin, N. *Eur J Immunol* 3(3): 159-164; 1977.

The development and growth of transplanted Lewis lung carcinoma into young and old syngeneic C57BL/6J mice were analyzed with respect to aging and changes in cell-mediated immune responses. Various features of cell-mediated immunity were assayed, including graft-vs-host (GvH) capacity, mixed lymphocyte culture (MLC) reactivity, and response to mitogens. It was found that the frequency and rate of tumor growth were increased when the tumors were implanted in older mice. To determine if this phenomenon was due to changes in the host immunologic system, spleen cells of mice of various ages were mixed with tumor cells and injected into recipient mice. It was found that active T cells from older mice increased tumor growth in the recipient mice. The results suggest that the process of aging is linked with a progressive increase in the proportion of immature T cells. An immunologic enhancing effect of a certain T cell subpopulation was suggested as the cause of tumor growth stimulation. (26 refs.)

7-2234 **Immunoprophylaxis in Lewis Lung Tumor with Vitamin A + BCG.** (Eng.) Kurata, T. (Inst. Cancer Res., Univ. Vienna, Austria) Micksche, M. *IRCS J Surg Sci: Cancer* 5(6): 277; 1977.

Experiments with male B6D2F1 mice with Lewis lung tumors indicated that there was a significant decrease in lung metastases in mice treated with vitamin A plus BCG. The mechanisms involved in the combined therapy are unclear. (refs.)

7-2235 **Alterations in the Cell-Surface Membrane of the Intestinal Epithelial Cell During Mitosis and Differentiation, and after Neoplastic Transformation.** (Eng.) Weiser, M. M. In: *Membranes and Diseases*. Bolis, L.; Hoffman, J. F.; Leaf, A., eds. (New York: Raven Press): pp. 289-290; 1976.

Isolated rat intestinal villus and crypt cells, human fetal intestinal cells, and rat intestinal 1,2-dimethylhydrazine-induced tumor cells were evaluated for the presence of glycosyltransferase activities and the corresponding glycoprotein receptors on their cell surface. The fetal intestinal epithelial cell, the mitotically undifferentiated crypt cell, and the intestinal tumor cell appeared to have on their cell surface glyco-

syltransferases as well as incomplete glycoproteins, which could serve as endogenous acceptors for these surface enzymes. These factors were largely absent in the mature villus epithelial cell-surface membrane. Rat small intestinal tumor cells also showed increased susceptibility to agglutination by concanavalin A (Con A). There appears to be a correlation among incomplete glycoprotein on the cell surface, glycosyltransferase activity, and Con A agglutination. These properties of the cell surface seem to be indicative of the undifferentiated mitotically active or dedifferentiated cell. (35 refs.)

77-2236 **The Role of Cell-Surface Sialic Acid in Lectin-induced Agglutination of Novikoff Hepatoma Cells.** (Eng.) Neri, G.; Giuliano, M. C.; Hixson, D. C.; Walborg, E. F. In: *Membranes and Disease*. Bolis, L.; Hoffman, J. F.; Leaf, A., eds. (New York: Raven Press): pp. 163-171; 1976.

The effect of neuraminidase (which releases sialic acid from the cell surface) on the agglutination of Novikoff hepatoma cells by concanavalin A (Con A), wheat germ agglutinin (WGA), *Ricinus communis* agglutinin I (RCAI), RCAII, and soybean agglutinin (SBA) was investigated to determine the role of cell-surface sialic acid in lectin-induced agglutination. Each of the lectins possesses different saccharide specificities: Con A for  $\alpha$ -D-glucosyl- or  $\alpha$ -D-mannopyranosyl residues; WGA for 2-acetamido-2-deoxy-D-glucose residues; RCA for D-galactopyranosyl residues; RCAII for D-galactosyl- and 2-acetamido-2-deoxy-D-galactopyranosyl residues; and SBA for 2-acetamido-2-deoxy-D-galactopyranosyl residues. The most prominent effect of removal of sialic acid from the cell surface was a marked increase in agglutination by RCAII and SBA. Neuraminidase digestion produced only a 2-fold increase in agglutination of Novikoff cells by RCAI, but a 12-fold increase by RCAII. Since both RCAI and RCAII possess specificity for D-galactosyl residues and RCAII also possesses specificity for 2-acetamido-2-deoxy-D-galactose, it is suggested that neuraminidase altered the expression of 2-acetamido-2-deoxy-D-galactose at the cell surface. This hypothesis was confirmed by the fact that neuraminidase-treated Novikoff cells were highly agglutinated by SBA, a lectin that demonstrates high specificity for 2-acetamido-2-deoxy-D-galactose. (29 refs.)

\* (Review): 77-1824, 77-1825, 77-1826, 77-1827, 77-1828, 77-1829, 77-1830, 77-1831, 77-1832, 77-1833, 77-1834.

\* (Chem): 77-1942.

\* (Phys): 77-1966, 77-1997.

\* (Viral): 77-2016, 77-2048, 77-2049, 77-2050.

\* (Path): 77-2237, 77-2239, 77-2240, 77-2262, 77-2273.



## PATHOGENESIS

- 77-2237 **Alpha-Chain Disease: Evidence for Common Clonal Origin of Intestinal Immunoblastic Lymphoma and Plasmacytic Proliferation (Letter to Editor).** (Eng.) Brouet, J. C. (Lab. Immunochemistry and Immunopathology--INSERM U 108, Res. Inst. on Blood Diseases, Hopital Saint-Louis, Paris 10, France) Mason, D. Y.; Danon, F.; Preud'homme, J. L.; Seligmann, M.; Reyes, F.; Navab, F.; Galian, A.; Rene, E. *Lancet* 1(8016): 861; 1977.

Direct evidence supporting the hypothesis that the malignant lymphomas that may arise in the late course of alpha-chain disease ( $\alpha$ -CD) are derived from the same B-cell clone as the initial plasma cell proliferation is presented. A 37-yr-old Iranian woman was diagnosed as having  $\alpha$ -CD, and a 9-cm jejunal segment showing the usual plasmacytic proliferation was resected. The patient received monthly courses of prednisolone and melphalan, followed by weekly treatments with cyclophosphamide and oral tetracycline. The patient remained symptom-free for about 2 yr. At that time, the  $\alpha$ -CD protein had almost completely disappeared from the serum but was still found in the jejunal juice. A barium meal revealed a large ulcer at the duodenojejunal junction, and a 5-cm ulcerated tumor was resected. Histologic examination showed that the tumor was composed of large lymphomatous cells with immunoblastic features, with exceptional Reed-Sternberg-like cells and some mature plasma cells with transitional forms between the latter and the large lymphoma cells. Immunofluorescence studies revealed that the intracytoplasmic  $\alpha$  chains were found only in the rare mature-looking plasma cells. A high density of  $\alpha$  chains was discovered on the surface of the large immunoblastic cells. These findings support the hypothesis of a common clonal origin for the large immunoblastic lymphoma cells and the plasmacytic cells. (8 refs.)

- 77-2238 **Pseudolymphomatosis of the Stomach.** (Rus.) Kurkin, A. V. (Dept. Pathological Anatomy, Abu Ali Ibn-Sina Tadjik Medical Inst., Dushanbe, USSR) Krakov, E. A. *Arkiv Patol* 39(1): 58-60; 1977.

A 43-yr-old man was first seen with chronic gastritis; 3 yr later, x-rays showed stomach cancer, and a laparotomy was performed. Pathologic findings of the tumor, which had infiltrated the pyloric region of the stomach, the duodenum, the hepatoduodenic ligament, and the head of the pancreas, were interpreted as lymphogranulomatosis of the stomach. He was treated with hormones and chemotherapy (prednisone, vinblastine, Leukan, bruneomycin), and for 5 yr he was in good health; however, his condition then worsened and he died. At autopsy, a 12- × 8.5- × 2.5-cm mass was found at the lesser curvature of the stomach; the histology of the tumor (which had infiltrated the muscle layer) and of the liver

and retroperitoneal metastases was mucinous carcinoma. The possibility that the patient had the poorly differentiated tumor at the time of palliative surgery was ruled out. Reexamination of the biopsy specimen showed that, initially, the patient had polymorphic cell pseudolymphogranulomatosis, which was misdiagnosed as lymphogranulomatosis. (3 refs.)

- 77-2239 **Immunological Depletion Contributing to Familial Hodgkin's Disease.** (Eng.) McBride, A. (St. Vincent's Hosp., Elm Park, Dublin 4, Ireland) Fennelly, J. J. *Eur J Cancer* 13(6): 549-554; 1977.

The occurrence of Hodgkin's disease in three sisters of a sibship of five girls over a period of 6 yr provided an ideal situation for the investigation of possible etiological factors. Of note was the occurrence of recurrent herpes labialis in the fourth girl, infectious mononucleosis in the fifth sister and recent herpes zoster in the mother. The main findings were (1) a familial depletion of T-lymphocyte function, as evidenced initially in all family members by depressed lymphocyte transformation, and (b) the presence of HL-A 1,8 in two affected sisters, the two unaffected sisters, and the mother, who had, however, transmitted these antigens in the normal fashion. Of significance was consistent skin anergy to dinitrochlorobenzene and tuberculin in the mother (HL-A 1,8) and in the fourth daughter (HL-A 1,8), even 2.5 yr after she had glandular fever. In addition, the mother, who has no outward signs of disease, had a paraprotein in her serum that formed almost 60% of the gammaglobulin fraction. It is concluded that a maternally transmitted immunologic depletion of T lymphocytes rendered the daughters vulnerable to infection by an oncogenic virus or an allied environmental agent. (15 refs.)

- 77-2240 **Polymorphonuclear Function in Acute Myeloblastic Leukemia.** (Eng.) Coiffier, B. (Service des Maladies du sang, Pavillon E bis, Hopital Edouard-Herriot, 69374, Lyon Cedex 2, France) Frobert, Y.; Revol, L. *Biomedicine* 27: 94-96; 1977.

A study of granulocyte functions in 15 patients with acute myeloblastic leukemia is reported. The functions were assessed by the ability of polymorphonuclear cells to migrate in Boyden's chamber and to ingest and kill *Staphylococcus aureus*. Chemotaxis was grossly impaired. Cellular impairment of phagocytosis and bactericidal capacity was observed in five and nine patients, respectively. Inhibitors of phagocytosis and bactericidal capacity were present in nine and four patients, respectively. The results improved in complete remission. (16 refs.)

- 77-2241 **Cancer Risk of the Stomach Resected for Ulcer: The Role of Duodenogastric Reflux.** (Eng.) Dahm, K. (Dept. Surgery, Univ. Hamburg, Martiastr. 52, D-2000 Hamburg 20, W. Germany) deHeer, K. *Z Krebsforsch* 87(3): 343-344; 1977.

A study was conducted to determine which type of gastroenteric anastomosis is predisposed to the development of carcinoma of the gastric stump. A total of 74 patients with carcinomas of the gastric stump following ulcer resection were evaluated. A Billroth II resection with retrocolic anastomosis had been performed in 64 patients, a Billroth II resection with anteroanastomosis in 7, and a Billroth I in 3. The frequency of carcinoma in the patients with a Billroth II resection with retrocolic anastomosis was about 9:1 compared to that in patients with an antecolic anastomosis. (1 ref.)

- 77-2242 **Tissue Culture, Chromosomal and Ultrastructural Features of Human Gastrointestinal Polyps (Meeting Abstract).** (Eng.) Natarajan, M. (Beth Israel Medical Center, New York, NY 10003) Mitra, J.; Stenger, R. J. *Fed Proc* 36(3): 1075; 1977. (no refs.)

- 77-2243 **An Analysis of Distant Metastasis from Squamous Cell Carcinoma of the Upper Respiratory and Digestive Tract (Meeting Abstract).** (Eng.) Merino, O. M. D. Anderson Hosp., Dept. Radiotherapy, Houston, TX) Lindberg, R. D.; Fletcher, G. H. *Int J Radiat Oncol Biol Phys* (Suppl 1): 85; 1976. (no refs.)

- 77-2244 **Gastrointestinal Cancer Studies in the Human to Nude Mouse Heterotransplant System.** (Eng.) Schmidt, M. (Memorial Sloan-Kettering Cancer Center, New York, NY) Deschner, E. E.; Thaler, H. T.; Clements, L.; Good, R. A. *Gastroenterology* 72(5, part 1): 829-837; 1977.

A report is presented of the establishment in nude mice and subsequent growth characteristics of human gastric adenocarcinoma, gastric leiomyosarcoma, histiocytic lymphoma of the stomach and gallbladder, pancreatic adenocarcinoma, colonic cancers and cell lines of duodenal (HUTU-80) and pancreatic (HS-766-T) cancers; melanoma (SK-Mel-5) and the murine metastasizing Lewis lung carcinoma. The rate of successful xenografting varied from 100% with colonic and duodenal cancer to 50% for a pancreatic cancer to only 17% for gastric adenocarcinoma. Pancreatic and colonic adenocarcinomas were maintained by successive xenotransplantation over 16 and 19 mo, respectively. Human xenografts retained a morphological identity with the tissues of origin through several transplant generations, but they did not metastasize. It is suggested that the nude mouse model can

be used to evaluate the endogenous properties of gastrointestinal cancers and their responses to exogenous agents. (64 refs.)

- 77-2245 **Spatiotemporal Development of Walker's Tumor Implanted in the Gastric Wall of Wistar Rats.** (Fre.) Santini, R. (Laboratoire de Physiologie et de Pharmacodynamie, I.N.S.A., 20 avenue Albert Einstein, Bat. 406, 69621 Villeurbanne, France) Brard, E. *C R Soc Biol* 170(6): 1239-1242; 1976.

Morphological and histological aspects of tumor growth in Wistar rats after implantation of Walker carcinoma 256 cells in the antral wall of the stomach are reported. A suspension of the tumor (2 mg/ml Ringer's solution; 30,000-50,000 cells/ $\mu$ l soln) was prepared from a Walker tumor implanted 8 days previously in a rat, and 0.1 ml was implanted using a fine intradermal needle in the stomach of the anesthetized recipient rat. The rats were sacrificed at different stages of tumor development over a 16-day period. Tumor growth was slow during days 1-9 with localization within the submucosa. The tumor growth rate accelerated on days 9-16, with increasing disorganization of the anatomy of the antrum, and death occurred on days 12-16. Histologically the tumor was a medullary-type carcinoma with the tumor parenchymal cells grouped in islands separated by scanty stroma. (2 refs.)

- 77-2246 **Pancreatic Cancer in Father and Son (Letter to Editor).** (Eng.) Reimer, R. R. (Environmental Epidemiology and Medicine Branches, NCI, Bethesda, MD 20014) Fraumeni, J. F.; Ozols, R. F.; Bender, R. *Lancet* 1(8017): 911; 1977.

The occurrence of a pancreatic tumor in successive generations is reported. A 63-yr-old chemical worker and his 36-yr-old son both developed pancreatic cancer. Several years before he died, the son worked briefly with his father, and both were exposed to vinyl chloride and other chemicals. Neither patient smoked or had any condition that predisposes to pancreatic cancer. The patients consumed large quantities of pistachio nuts, and they may have ingested some that were contaminated with mycotoxins. The occurrence of this tumor in successive generations may be due to chance or genetic transmission. Some characteristics of the case suggest an environmental influence. (8 refs.)

- 77-2247 **Unique Features of Serially Transplanted Human Pancreatic Cancer in Nude Mice.** (Eng.) Kim, D. K. (Dept. Surgery, Memorial Sloan-Kettering Cancer Center, New York, NY) Kakita, A.; Cubilla, A.; Fleisher, M.; Fortner, J. G. *Surg Forum* 27: 142-144; 1976.



Adenocarcinoma of the pancreas were successfully transplanted into 158 nude mice over a 15-mo period, and their neoplastic features were examined by comparing the findings of each transplant generation to the original tumor as well as to previous generations. Pancreatic adenocarcinomas were obtained originally from two patients who underwent pancreatectomy. Carcinomatous tissue was diced into 1- × 1-mm pieces by a sterile technique. Minced tumor was soaked slightly into Hank's soln. Approx 5 × 5 mm of minced tumor was then transplanted sc into the lateral chest wall of 6-wk-old nude mice (Swiss Nu/Nu). Tumor tissue for serial passage was obtained from a healthy tumor-bearing mouse at each generation. Each of the two tumor lines exhibited persistent differences in growth pattern. Overall, the growth rate was rapid (tumor size was 280 mm<sup>3</sup> by the eighth week), and the incidence of tumor graft take was high (100% by the eighth week) in one line. Growth was slow (66 mm<sup>3</sup>) and incidence of graft take was lower (63%) in the other. Median survival times of host animals were 9 and 10 wk for each of the two tumor lines. Those patterns were similar in each transplant generation. Light and electron microscopic studies revealed minimal deviation from the original human tumor characteristics. Tumor markers remained intact. Tumor carcinoembryonic antigen (CEA) levels were 22,000 nanograms (ng)/g of wet tumor for the first transplant generation and 5,333 ng/g in the fifth generation for one tumor line. For the other, CEA levels of 45,000 ng/g were found in the first transplant generation and 8,783 ng/g in the seventh. Plasma CEA levels had been normal in both patients. CEA levels in the plasma of tumor-bearing nude mice were elevated slightly in some animals. No tumor metastasis was found in nude mice growing carcinoma. Results show persistence of neoplastic characteristics of human pancreatic cancer 15 mo after serial transplantations in nude mice. The exception is the loss of the ability to metastasize. (5 refs.)

- 77-2248 **Alpha-1-Antitrypsin and Hepatocellular Carcinoma.** (Eng.) Palmer, P. E. (Dept. Pathology, New England Medical Center Hosp., Boston, MA 02111) Wolfe, H. J. *IRCS Med Sci: Cancer* 5(3): 109; 1977.

Tissue and serum samples from 10 patients with hepatocellular carcinoma were studied to correlate serum phenotype with tissue deposits of  $\alpha$ -antitrypsin (AAT). Tissue deposits of AAT were determined using PAS staining and immunoperoxidase tests. Protease inhibitor (Pi) phenotyping was accomplished by starch gel electrophoresis and crossed immunoelectrophoresis. The MM phenotype is associated with normal serum AAT levels and the ZZ phenotype is associated with low serum AAT levels. MZ phenotypes were found in 3/10 hepatoma patients, and in another 4 cases unexplained extra electrophoretic bands that interfered with interpretation were present. This problem was not encountered with the control group. The three MZ phenotypes represent an excess, by a factor > 10, over the number expected, as determined from blood donor population data. The findings suggest that the MZ phenotype is associated with

hepatocellular carcinoma and that it may represent a genetically determined risk factor. (7 refs.)

- 77-2249 **Early Lesions and Development of Primary Hepatocellular Carcinoma in Man** (Meeting Abstract). (Eng.) Okita, K. (First Dept. Internal Medicine, Yamaguchi Univ. Sch. Medicine, Ube, Japan) Kodama, T. *Gastroenterol Jpn* 12(1): 83; 1977. (5 refs.)

- 77-2250 **Clinicopathological Study Concerning the Development of Hepatocellular Carcinoma on the Basis of Chronic Liver Disease** (Meeting Abstract). (Eng.) Obata, H. (Inst. Gastroenterology, Tokyo Women's Medical Coll., Tokyo, Japan) Tamiya, M. *Gastroenterol Jpn* 12(1): 81; 1977. (no refs.)

- 77-2251 **Development of Hepatocellular Carcinoma (HCC) During the Follow-up Study of Chronic Liver Diseases; Clinical Observation in 24 Cases** (Meeting Abstract). (Eng.) Kubo, Y. (Second Dept. Medicine, Kurume Univ. Sch. Medicine, Kurume, Japan) Yakushiji, F. *Gastroenterol Jpn* 12(1): 82; 1977. (no refs.)

- 77-2252 **Morphological Studies on the Course from Hepatitis to Hepatoma** (Meeting Abstract). (Eng.) Ishiguro, Y. (Third Div., Dept. Internal Medicine, Niigata Univ., Niigata, Japan) Ichida, F. *Gastroenterol Jpn* 12(1): 81-82; 1977. (1 ref.)

- 77-2253 **Preneoplastic and Collagenizing Changes in the Liver and Blood Clotting in PVC Workers.** (Ger.) Bachner, U. (No affiliation given) Etzel, F.; Muller, N.; Lange, C. E.; Egli, H. *Thromb Diath Haemorrh Suppl* 62: 319-326; 1976.

Sixty-five polyvinyl chloride (PVC) workers with thrombocytopenia were divided into three groups: (A) 31 with unremarkable liver histology; (B) 20 with slight liver fibrosis; and (C) 14 with clear periportal and perisinusoidal fibrosis, stellate (Kupffer) cell proliferation, and atypical stellate cells. Twenty-two blood-clotting tests were carried out and related to pathological and histological findings in the liver. PVC workers with strong cirrhotic and preneoplastic changes in the liver showed the lowest number of thrombocytes. Groups B and C showed an increase in platelet factor 3 activity, but this increase was statistically significant in Group C only. Factor VIII activity showed a tendency to rise from Groups

A to C. Factor V values were normal in Groups A and B, but slightly decreased in Group C. In all three, fibrinogen was at high normal or elevated levels, plasminogen was at the lower level of normal, and aggregability was increased. The reaction time in TEG was slightly shorter in Group C than in A or B. Tissue changes in the liver and spleen were related to hindrance of the microcirculation, which could explain the portal hypertension and esophageal varices observed in 10/14 Group C patients. The availability of phospholipids was increased, as shown by the deposit of lipopigments in the sinusoidal and liver cells. (45 refs.)

**7-2254 Glioblastoma Multiforme: Determinants of Time-Dose-Volume Factors (Meeting Abstract).**

(Eng.) Salazar, O. M. (Div. Radiation Oncology, Univ. Rochester Cancer Center, 601 Elmwood Avenue, Rochester, NY) Rubin, P. *Int J Radiat Oncol Biol Phys* 1(Suppl 1): 28; 1976. (no refs.)

**7-2255 Heterotransplantation of Human Glioblastoma Multiforme and Meningioma to Nude Mice.**

(Eng.) Rana, M. W. (Dept. Anatomy, Saint Louis Univ. Sch. Medicine, 1402 S. Grand Boulevard, St. Louis, MO 63104) Pinkerton, H.; Thornton, H.; Nagy, D. *Proc Soc Exp Biol Med* 155(1): 85-88; 1977.

The successful sc transplantation and growth of a human glioblastoma and a meningioma in male nude BALB/c mice, with retention of their unique histologic features in each case, are reported. (1) Ten 6-wk-old mice were inoculated sc with  $5 \times 10^6$  highly anaplastic cells from human glioblastoma T98; (2) 6 15-wk-old mice were implanted sc in the right dorsal area with 0.025 ml of a nonnecrotic portion of a human glioblastoma Br7 mince; (3) 8 8-wk-old mice were implanted sc in the lateral or mid-dorsal area with the same amount of human meningioma mince. None of the 10 T98 mice developed tumors, but 3/6 Br7 glioblastoma mice developed tumors 2 mo after implantation and 5/8 meningioma mice developed tumors at 6-7 wk. Tumors of both xenografts were passed successfully to a second generation, with complete retention of their unique histologic features. Both tumor types contained scattered cytoplasmic A-type virus particles and immature and mature C-type particles (presumably representing a xenotropic mouse virus). The glioblastoma showed many neuroglia fibers. The growth of the two tumors in nude mice may be a useful model for etiologic, immunologic, and chemotherapeutic studies. (6 refs.)

**7-2256 Di George Syndrome Associated with Glioma and Two Kinds of Viral Infection (Letter to Editor).** (Eng.) Asamoto, H. (Natl. Kyoto Hosp., Fushimiku, Kyoto, Japan) Furuta, M. *N Engl J Med* 296(21): 1235; 1977.

A case of Di George's syndrome associated with glioma occurred in an infant girl whose birth wt was 3,150 g. Admission was at 20 days of age, and the patient presented with fever and generalized clonus lasting 5-10 sec and occurring 40-80 times a day. She died of respiratory failure at the age of 46 days. Autopsy revealed a ventricular septal defect; absence of the thymus and parathyroid glands; enlargement of the mesenteric, inguinal, and axillary lymph nodes; splenomegaly; and hepatomegaly. Cytomegalovirus was identified in the lungs, adrenal glands, lymph nodes, small intestine, and spleen. ECHOvirus was isolated from the small intestine and brain. Well-circumscribed gliomas were discovered in the paraventricular area of the cerebrum. The lack of T cells in this patient may have rendered her susceptible to the development of neoplasia possibly caused by an oncogenic virus. (4 refs.)

**77-2257 Primary Germinal Brain Tumors.** (Fre.) Juif, J. C. (Service de Pédiatrie III, C.H.U., 67000 Strasbourg, France) Maitrot, D.; Pierson, M.; Heldt, N.; Buchheit, F.; Luckel, J. *Cl. Arch Fr Pédiatr* 34(4): 335-346; 1977.

Four case histories are used to illustrate cerebral tumors of primary germ cell origin, a histological diagnosis new to the French medical literature. The tumors appear most often in young adult men, and they involve the pineal body, the walls of the third ventricle, the hypothalamus, and the posterior pituitary. Clinically, the first stage is of long duration and is characterized by endocrine disorders, particularly water balance; neurological symptoms appear in the second stage. In the first case, a 9-yr-old girl, the tumor was not suspected until shortly before death, when surgery revealed a voluminous tumor occupying all of the third ventricle and infiltrating the hypothalamus and the infundibulum. On histological examination, the tumor had numerous cells of varied size with a clear cytoplasm, atypical nuclei, and frequent mitoses; conjunctive tissue with many islands of lymphocytes was abundant. The second case, a 10-yr-old boy, also died subsequent to surgery. He had a tumor comprised of smooth muscle and cartilaginous elements mixed with lung tissue. Two 20-yr-old men had tumors characterized by plasma cell and lymphocytic infiltration and rich vascularization. Radiotherapy alone (6,500 and 7,000 rads along the cerebrospinal axis) was used in these patients, and their symptoms regressed after treatment. (35 refs.)

**77-2258 Retinal Neoplasia and Dysplasia. II. Retinoblastoma Occurring with Persistence and Hyperplasia of the Primary Vitreous.** (Eng.) Irvine, A. R. (Howe Lab. Ophthalmology, 243 Charles St., Boston, MA 02114) Albert, D. M.; Sang, D. N. *Invest Ophthalmol Visual Sci* 16(5): 403-407; 1977.



A case of retinoblastoma in an eye with persistent hyperplastic primary vitreous (PHPV) in an infant boy is reported. At birth, the boy had right microphthalmus and a small tumor of the scalp in front of the anterior fontanelle. The lesion was excised at 5 mo of age, and microscopically, it was found to be consistent with metastatic neuroblastoma. The child received four doses of x-rays to the scalp lesion. An exploratory laparotomy was performed at 10 mo of age, but it failed to reveal tumor. During the following 8 mo the scalp lesion continued to grow, and a lesion appeared in the left eye. A second biopsy of the scalp lesion was diagnosed as metastatic retinoblastoma to the scalp from the right eye. At 18 mo, the right eye was enucleated. The patient was then treated with Cytosan, 5 mg/kg body wt/day every 2 wk for 10 wk. This was followed by 300 kilorads of x-rays to the brain and 4,800 rads to the skull. A swelling of the right mandible that developed 4 mo later was treated with 4,351 rads of cobalt-60. Bone marrow studies were negative, and the child was discharged on Cytosan therapy. The patient died 2 mo later at approx 3 yr of age. An autopsy was not performed. (3 refs.)

**77-2259 "Brown Tumor" of Hyperparathyroidism Induced with Anticonvulsant Medication.** (Eng.)

Campbell, J. E. (Sunnybrook Medical Centre, Univ. Toronto, 2075 Bayview Ave., Toronto, Ontario M4N 3M5, Canada) Tam, C. S.; Sheppard, R. H. *J Can Assoc Radiol* 28(1): 73-76; 1977.

A case history is presented of a 50-yr-old epileptic who had been receiving large doses of phenobarbital (phenylethylmalonylurea, 460 mg/day) and small doses of dilantin (diphenylhydantoin, 100 mg bid). A brown tumor of hyperparathyroidism was discovered and subsequent investigation revealed secondary hyperparathyroidism and osteomalacia. (15 refs.)

**77-2260 Primary and Secondary Carcinomata with Focal Nodular Hyperplasia in A Multinodular Thyroid: Case Report.** (Eng.) Kim, E. (Div. Nuclear Medicine, Washington Univ. Sch. Medicine, 510 S. Kings-highway Blvd., St. Louis, MO 63110) Mattar, A. G. *J Nucl Med* 17(11): 983-984; 1976.

The case history of a 57-yr-old woman with a multinodular thyroid gland is presented. Surgery was performed and the right lobe and isthmus were resected. Histological examination of the surgical specimens showed focal hyperplasia involving the nodule from the right upper pole, metastatic renal-cell carcinoma in the isthmus nodule, and papillary adenocarcinoma of the thyroid in a small nodule not detected clinically. In patients with malignant disease, the development of recent palpable or scan abnormalities in the thyroid should raise the possibility of metastases. (6 refs.)

**77-2261 Oncocytic Neoplasms of Salivary Glands: An Ultrastructural Study.** (Eng.) Johns, M. E.

(Otolaryngology Service, Walter Reed Army Medical Center, Washington, DC 20012) Regezi, J. S.; Batsakis, J. G. *Laryngoscope* 87(6): 862-871; 1977.

The electronic microscopic (EM) features of three oncocytic carcinomas (in 3 men aged 49, 61, 80 yr) and two benign oncocytomas (a 56-yr-old woman and a 72-yr-old man) of the salivary glands are described, and the clinical presentation, treatment, and biologic course of these rare tumors are included. Although there are limitations in the use of tissue removed from paraffin for EM, the mitochondria could be recognized readily, and identification of oncocytes was reliable. The cells of the oncocytic carcinomas were characterized by an unusually large number of mitochondria that also showed increased size, pleomorphism, and atypism. They contained few other subcellular organelles and no myofilaments or glycogen, as described previously in the benign tumors. Although EM is an adjunct to correct pathologic diagnosis, it does not determine whether an oncocytic tumor is malignant or not. Diagnosis of malignancy must be based on clinical or histological evidence of local invasion or metastases. Oncocytes demonstrate high oxidative activity on histochemical staining. The stains are of value in making a diagnosis of oncocytoma when positive, but they are not of value in excluding the diagnosis when negative. (17 refs.)

**77-2262 Breast Cancer Arising in Surgical Scars.** (Eng.)

Freund, H. (Hadassah Univ. Hosp., Post Office Box 499, Jerusalem, Israel) Biran, S.; Laufer, N.; Eyal, Z. *J Surg Oncol* 8(6): 477-480; 1976.

Between 1961 and 1975, 12 women (27-72 yr old, av 42 yr) with breast cancer arising at the site of an old surgical scar were observed. In nine patients the left breast was involved and in three the right, with the outer, upper quadrant predominating. Six women developed breast cancer at the site of scars from former biopsies for benign lesions performed 2-30 yr (av 10.5 yr) prior to detection of the present cancer. Three women had one or more breast abscesses drained during lactation periods 12-23 yr (av 17 yr) earlier. Three other patients developed breast cancer within a thoracotomy scar reaching the breast. The thoracotomy had been performed for tuberculosis in one woman and for closed mitral valvotomy in two patients 6 and 9 yr previously. All tumors were operable. Ten women had a radical mastectomy, 1 a simple mastectomy, and 1 a "lumpectomy" because of a severe heart condition. In 10 cases the carcinoma was of the scirrhous type; in the remaining 2 patients inflammatory and anaplastic carcinomas, respectively, were noted. The combination of trauma as an oncogen and scar tissue as a functional and immunological site of decreased resistance appears to play a major role in the development of breast cancer. (15 refs.)

**77-2263 Concentration of Testosterone Glucuronide in Urine from Women with Breast Tumours.**

(Eng.) Jones, M. K. (Faith Courtauld Unit Human Studies Cancer, King's Coll. Hosp. Medical Sch., London SE5 8RX, England) Ramsay, I. D.; Collins, W. P. *Br J Cancer* 5(6): 885-887; 1977.

The concentration of testosterone glucuronide was measured in 89 women, aged 25 to 70 yr, with either benign or malignant breast tumors. There was no significant difference in the concentration of the glucuronide in these patients either before or after menopause. (19 refs.)

77-2264 **Tumor Cells in Venous Blood Draining Mammary Carcinomas.** (Eng.) Golinger, R. C. (Inst. Pathology, Shadyside Hosp., 5230 Centre Ave., Pittsburgh, PA 15232) Gregorio, R. M.; Fisher, E. R. *Arch Surg* 112(6): 707-708; 1977.

Tumor cells were identified in mammary venous blood in 10/38 (26.3%) patients who underwent total mastectomy with axillary dissection performed as the primary therapy for breast carcinoma (pathological stages I and II). They appeared as solitary cells in all cases except two, in which they were clumped. An attempt was made to correlate this phenomenon with 35 histological characteristics of the primary tumors. The presence of blood-borne tumor cells was inversely related to perineural space invasion, whereas there was no significant relationship with the remaining 34 characteristics. The frequency and significance of direct hematogenous dissemination of tumor cells during mastectomy remain unknown. (9 refs.)

77-2265 **The Relationship of Gross Cystic Disease of the Breast and Carcinoma.** (Eng.) Haagensen, C. D. (Columbia Presbyterian Medical Center, New York, NY) *Ann Surg* 185(3): 375-376; 1977.

A new classification for benign epithelial breast tumors has enabled some 5,000 lesions to be correlated with a long-term follow-up. The results show that patients with gross cysts (3 mm or more in diameter and forming a palpable tumor) develop breast carcinoma at approx four times the expected frequency. Two other lesions that clearly dispose to breast carcinoma are multiple intraductal papilloma and lobular hyperplasia (lobular carcinoma in situ). (no refs.)

77-2266 **Morphological Studies in Human Proliferating Mammary Tissues under Treatment with the Prolactin Inhibitor Bromocryptine (CB 154) (Meeting Abstract).** (Eng.) Zippel, H. H. (Universitäts-Frauenklinik Köln, Cologne, W. Germany) Schulz, K. D.; Del Pozo, E. *Acta Endocrinol [Suppl] (Kbh)* 84(208): 42; 1977. (no refs.)

77-2267 **Tubular Carcinoma of the Breast: Clinical, Histological, and Ultrastructural Observations.** (Eng.) Tobon, H. (Dept. Pathology, Magee-Womens Hosp., Sch. Medicine, Univ. Pittsburgh, PA 15213) Salazar, H. *Arch Pathol Lab Med* 101(6): 310-316; 1977.

Nine cases of primary unilateral tubular carcinomas of the breast were studied, and ultrastructural correlations were made in three. All of the patients (32-59 yr old) had small tumors (mean diameter, 1.2 cm), and in all the initial complaint was a palpable mass. Two patients underwent radical mastectomy, two had a modified radical mastectomy, and two had a simple mastectomy. Three patients had an excisional biopsy, one followed by radiation. Only 1/6 patients had residual tumor in the mastectomy specimens, and all are alive 5-68 mo after treatment. All biopsy specimens showed infiltrating angular tubules with single-lining epithelium, minimal anaplasia, rare mitosis, and scant or absent myoepithelium. The tumor cells were connected to each other by desmosomes, cytoplasmic interlocking processes, and tight junctions. The characteristic histological appearance of the stroma, dense and fibrotic around the neoplastic tubules, provides an important factor for differential diagnosis. A modified simple mastectomy or another conservative surgical therapeutic approach should suffice for the treatment of tubular carcinoma, considering its low incidence of spread. (15 refs.)

77-2268 **Feminizing Adrenocortical Carcinoma in a 66-Yr-Old Man.** (Ger.) Wirth, T. (Pathologisches Institut, Kantonsspital, CH-8401) Maurer, R.; Kappeler, H.; Krampf, H. *Schweiz Med Wochenschr* 107(12): 411-417; 1977.

A 66-yr-old man presented with bilateral gynecomastia, slightly elevated plasma cortisol and plasma testosterone levels, raised estrogen levels, and a space-forming process at the upper pole of the right kidney. The tumor was a necrotic, partially cystic, adrenal cortex carcinoma. Tumor recurrence occurred 1 yr later with possible growth into the kidney. Electron microscopy of the recurrent tumor showed that it developed from the fasciculoreticular layer. (17 refs.)

77-2269 **Origin of Embryo-derived Yolk Sac Carcinomas.** (Eng.) Damjanov, I. (Univ. Connecticut Health Center, Farmington, CT 06032) Skreb, N.; Sell, S. *Int J Cancer* 19(4): 526-530; 1977.

The origin of yolk sac carcinoma (YSC) obtained from rat embryos transplanted to extrauterine sites was determined. Portions of 9-day embryos from Lewis or Fisher rats transplanted underneath the kidney of isogenic males gave rise to well-differentiated teratomas. Histologic sections failed to show yolk sac epithelium, and serum  $\alpha$ -fetoprotein (AFP) concentrations were < 60 nanograms (ng) in all rats. Trans-



plants of extraembryonic portions of 9-day embryos could not be recovered or identified 7 wk after grafting. Transplants consisting of embryonic portions with a small extraembryonic portion gave rise to teratomas with areas of YSC. When larger numbers of extraembryonic cells were injected into Lewis rats, nodules measuring 5-10 mm were recovered 7 wk later. Serum AFP concentrations in these animals ranged from 16 to 88 ng/ml. The results indicate that under the appropriate conditions, YSC originates from the extraembryonic portions of the rat egg cylinder. Possible factors involved in the survival and proliferation of yolk sac epithelium in extrauterine sites are discussed. (14 refs.)

- 77-2270 **Childhood Gonadoblastoma and Seminoma in a Dysgenetic Cryptorchid Gonad.** (Eng.) Mandell, J. (Dept. Urology, Univ. North Carolina Sch. Medicine, Chapel Hill, NC) Stevens, P. S.; Fried, F. A. *J Urol* 117(5): 674-675; 1977.

A case of gonadoblastoma and seminoma occurring in a genotypic male child with ambiguous genitalia is presented. The child was admitted at the age of 4 days for evaluation of the ambiguous genitalia. Physical examination showed low-set ears, hypertelorism, a large left flank mass, a right inguinal mass, a bifid empty scrotum, and penoscrotal hypospadias. At surgical exploration, a dysplastic left kidney with ureteral atresia was removed. The child was treated with testosterone, and a decision was made to rear the child as a male subject. Evaluation 4 yr later showed a normal right kidney, but a cystourethrogram revealed a Grade III right vesicoureteral reflux and the presence of a large mullerian duct remnant. At surgery a mullerian remnant resembling a primitive left fallopian tube, uterus, and cervicovaginal canal was removed along with an indeterminate left intraabdominal gonad. Histologic examination of the gonadal tissue revealed a dysgenetic gonad with gonadoblastoma and seminoma. No further therapy was instituted. (7 refs.)

- 77-2271 **Three Sisters with Gonadoblastoma.** (Eng.) Ionescu, B. (Inst. Endocrinology, Bucharest, Romania) Maximilian, C. *J Med Genet* 14(3): 194-199; 1977.

The case histories of three sisters with gonadoblastoma are presented. The women were 24, 22, and 18 yr of age, and all were admitted for primary amenorrhea. At the time of initial tests, all three sisters had increased secretions of estrogens and androgens. Gonadectomy caused the levels of these steroid hormones to fall, suggesting a gonadal origin. Histopathological diagnosis was gonadoblastoma. All three sisters had a 46,XY karyotype, suggesting that heredity may play a role in the genesis of this tumor. (29 refs.)

- 77-2272 **Unusual Gonadal Stromal Tumor of the Testis. Case Report with Ultrastructural Observations.** (Eng.) Evans, H. L. (Dept. Pathology, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77025) Glick, A. D. *Arch Pathol Lab Med* 101(6): 317-320; 1977.

An unusual gonadal stromal tumor of the testis in a 4-yr-old boy was examined ultrastructurally, and the features are reported. The patient was admitted with a 10-day history of left testicular mass and mild bilateral gynecomastia. The left testicle was firm, and it measured  $3.0 \times 3.5$  cm on physical examination. A left radical orchiectomy was performed 3 days after admission, and the postoperative course was uneventful. The boy was well 3 mo postoperatively, and his gynecomastia decreased to a barely noticeable level. The tumor cells were rich in cytoplasmic microfilaments, with localized dense areas of the type observed in smooth muscle cells, as well as intracytoplasmic lipid droplets. The cells lacked the abundant smooth endoplasmic reticulum usually noted in neoplastic and non-neoplastic Leydig and Sertoli cells. The tumor cells demonstrate significant similarities to the immature fusiform interstitial cells and the peritubular contractile cells in the normal testis. (19 refs.)

- 77-2273 **Latent Carcinoma of the Prostate in a 24-Yr-Old Man Receiving Cyclophosphamide and Azathioprine.** (Eng.) Elliott, G. B. (Royal Jubilee Hosp., 1900 Fort St., Victoria, BC V8R 1J8, British Columbia, Canada) Silverberg, D. S.; Dossetor, J. B.; Muir, C. S. *Can Med Assoc J* 116(6): 651-652; 1977.

The case is reported of a 24-yr-old man who died after receiving cyclophosphamide (800 mg iv) and azathioprine (100 mg/day, po) for progressive glomerulonephritis. Examination of the patient's prostate, which was inadvertently collected for a whole-prostate survey, revealed a microacinar clear-cell carcinoma. (3 refs.)

- 77-2274 **Choriocarcinoma in Mother and Child, Identified by Immunoenzyme Histochemistry.** (Eng.) Nieuwenhuijzen Kruseman, A. (Pathologisch Laboratorium, University Medical Centre, Wassenaarseweg 62, Leiden, the Netherlands) Van Lent, M.; Blom, A. H.; Lauw, G. P. *Am J Clin Pathol* 67(3): 279-283; 1977.

A case of metastatic choriocarcinoma, after term pregnancy, in mother and still-born child is described. The 20-yr-old woman had no prior history of abortion or molar pregnancy. An autopsy of the macerated hydropic still-born fetus revealed a  $12 \times 18$  mm necrotic tumor of the left kidney. Four weeks after delivery the mother bled vigorously and curettage was performed. No tumor was found in the placenta. Microscopic examination of the scrapings revealed abnormal trophoblastic cells; pelvic angiography documented the uterine

calization of the tumor. High gonadotrophin levels also supported the suspicion of a choriocarcinoma. The patient appeared to be free of tumor after a 4.5-mo treatment with methotrexate (25 mg/day; continuous infusion) and folinic acid (12 g/day; im). An indirect immunohistoperoxidase technique suitable for formalin-fixed and paraffin-embedded tissue was used to demonstrate gonadotropin activity in the tumors of mother and child; identical immunohistochemical patterns for gonadotropin activity indicated that both malignant growths were choriocarcinoma. Because of a positive Riechauer test it was assumed that the massive fetomaternal transfusion caused the intrauterine death, not the tumor. This case shows the value of immunoenzyme histochemistry as a method of diagnosing endocrine tumors. (15 refs.)

77-2275 **Uterine Leiomyoma with Hemangiopericytomatous Foci: Histogenetic Implications.** (Eng.) Moore, L. H. (Dept. Pathology, Vancouver General Hosp., Vancouver, British Columbia, Canada) *Am J Obstet Gynecol* 137(8): 891-892; 1977.

The case of a 28-yr-old patient with a uterine leiomyoma with hemangiopericytomatous foci is reported. The histologic features of the leiomyoma suggested a vascular origin. (10 refs.)

77-2276 **Canine Transmissible Venereal Tumor in Iran.** (Eng.) Ivoghli, B. (Dept. Pathobiology, Sch. of Veterinary Medicine, Pahlavi Univ., Shiraz, Iran) *Vet Pathol* 14(3): 289-290; 1977.

Transmissible venereal tumor, the first one to be reported in the canine population in Iran, occurred. An adult female was taken to a veterinary clinic to be killed because of persistent ulcerated vaginal lesion. Autopsy showed a multilobular verrucous ulcerated tumor of the ventral wall of the vagina and vestibule. The perineal skin also had numerous ulcerated nodules. The metastatic tumors in the dermis and cutis had the same morphology as the vaginal tumor. No attempt was made to transmit the tumor for confirmatory diagnosis, but comparative histological studies offered strong evidence that the cutaneous as well as the genital tumor were transmissible venereal type. (5 refs.)

77-2277 **Mouse Teratomas and Embryoid Bodies: Their Induction and Differentiation.** (Eng.) Iles, S. A. (Dept. Zoology, South Parks Road, Oxford, OX1 3PS, England) *J Embryol Exp Morphol* 38: 63-75; 1977.

Induction and differentiation of mouse teratomas and embryoid bodies were studied in C3H and 129/J mice. Teratomas were induced by the transfer of C3H and 129/J blastocysts (day 4 of pregnancy) and C3H egg cylin-

ders (days 7 and 9) to extrauterine sites. Ten percent of the 129/J blastocysts gave rise to teratomas in the testes of the adult hosts, but they failed to develop into tumors in the kidney. Thirty-six percent of C3H blastocysts formed testicular teratomas. The egg cylinders of the C3H embryos gave rise to teratomas in the testis and kidney. Tumors derived from both strains were histologically similar. Culture in medium for 4 days increased the incidence of teratoma formation with the 129/J blastocysts from 10% to 30%, but not with the C3H blastocysts. None of the three primary tumors derived from the 129/J blastocyst was transplantable. Four transplantable teratomas (teratocarcinomas) were derived from the C3H embryos, and embryoid bodies were derived from each line. The differentiative capacity of the teratocarcinomas was similar whether they were maintained as a solid tumor or as embryoid bodies. Both growth conditions seemed to select for cells with rapid growth, which may have restricted developmental capacities. (21 refs.)

77-2278 **Teratoma in a Wistar Rat.** (Eng.) Schardein, J. L. (Dept. Toxicology, Res. and Medical Affairs Div. Parke, Davis and Company, 2800 Plymouth Road, Ann Arbor, MI 48106) Fitzgerald, J. E. *Lab Anim Sci* 27(1): 114; 1977.

A teratoma which surrounded the left kidney is described for the first time in a spontaneously hypertensive, sexually normal male Wistar rat. (8 refs.)

77-2279 **Blood Formation in a Clonal Cell Line of Mouse Teratocarcinoma.** (Eng.) Cudennec, C. A. (Institut d'Embryologie, CNRS et College de France, 49bis, Ave. de la Belle-Gabrielle, 94130 Nogent-sur-Marne, France) Nicolas, J. F. *J Embryol Exp Morphol* 38: 203-210; 1977.

Pluripotent cells of a mouse teratocarcinoma clonal line were cultivated in submerged cultures. The first macroscopic differentiation in the cultures was hollow vesicles, which in most cases (52/94 cultures) rapidly became associated with blood islands. Various stages in the development of these blood islands could be observed. The evolution was similar to the mesodermal differentiation of the normal embryo yolk sac. Despite the healthy state of the tissues, the formation of Hb-containing cells stopped after a while. The mean life span of the blood islands was 6 days; endodermal vesicles persisted for > 50 days. Whether stem cells capable of further erythroid differentiation are present in the explants could not be determined from these observations. (18 refs.)

77-2280 **A Study on Malignant Cycle of Gastric Ulceration (Meeting Abstract).** (Eng.) Yasui, A. (Dept. Gastroenterological Surgery, Koshigaya City Hosp., Koshigaya, Saitama Prefecture, Japan) *Gastroenterol Jpn* 12(1): 92-93; 1977. (no refs.)



77-2281 Studies on the Cancerous Change from the Gastric Ulcer by Follow-up for Long Term with Endoscopy and Biopsy (Meeting Abstract). (Eng.) Oguro, Y. (Natl. Cancer Center Hosp., Tokyo, Japan) Yoshida, S. *Gastroenterol Jpn* 12(1): 91-92; 1977. (no refs.)

77-2282 Association of Cholelithiasis and Cancer of the Gastrointestinal Tract. (Fre.) Germain, M. (Laboratoire d'Anatomie Pathologique, Hopital Antoine Beclere, 157, rue de la Porte de Trivaux, F 92140 Clamart, France) Martin, E.; Gremillet, C. *Nouv Presse Med* 6(16): 1393; 1977.

The incidence of cholelithiasis associated with gastrointestinal cancers is reviewed and three brief case reports are presented. The three patients were women, ages 62, 72, and 74 yr. X-rays taken after patients were hospitalized for abdominal pain showed the presence of calculi in the gall bladders. Cholecystectomies revealed primary carcinoma of the gall bladder with metastases in 2/3 patients, and a polymorphous carcinoma of the colon was found in the third patient. Among 120 cases of cholelithiasis gathered from the literature, there were 24 with an associated cancer (13 of the biliary tract and 11 extrabiliary). It is concluded that patients with cholelithiasis should be examined for cancer metastases or primary cancer in the gastrointestinal tract, and, more important, all gall bladders with demonstrated calculi should be removed surgically. (no refs.)

77-2283 Histologic Changes in Patients with Gastric Carcinoma. Study of Cases with a Recurrence More than Five Years after Surgery. (Jpn.) Enjoji, M. (Second Dept. Pathology, Faculty Medicine, Kyushu Univ., Fukuoka, Japan) Koga, S. *Stomach Intest* 12(1): 33-40; 1977.

A description is given of the gross and microscopic characteristics of primary carcinoma of the stomach in 15 patients (8 men and 7 women aged 35 to 67 yr) in whom a recurrence developed > 5 yr after initial surgery. In 6/15, the recurrent tumor was also studied. Sections from 42 patients with a recurrence < 5 yr after surgery were studied for comparison. Compared to the surgically removed primary tumor, the

recurrent tumor had features indicating an exacerbation in growth, such as a higher number of atypical cells and nuclei, an increased growth of a poorly differentiated parenchyma, and a more extensive lymphatic and blood vessel permeation by the malignant cells. A marked lymphoid infiltration observed in the stroma of two patients had almost completely disappeared when the recurrent tumor was examined 7 yr later. The changes were more striking in patients with a late recurrence than in those with an early recurrence. (6 refs.)

77-2284 Histogenesis and Morphological Characteristics of Stomach Cancer. (Rus.) Dikshtein, E. A. (Dept. Pathoanatomy, M. Gorkii Donetsk Medical Inst., Donetsk, USSR) Vasilenko, I. V. *Arkhl Patol* 39(1): 11-17; 1977.

The etiology, epidemiology, and clinical features of two histologically different types of stomach cancer are discussed, and the results of comparative histochemical analyses of the tumor specimens from 24 patients with well-differentiated glandular adenocarcinoma of the stomach and from 51 patients with poorly differentiated carcinoma of the stomach are presented. Poorly differentiated carcinoma occurred more often among patients < 50 yr (32/51) compared to those with glandular carcinomas (5/24 patients were < 50 yr). Alkaline phosphatase activity was practically the same in both types of carcinoma. Marked secretion of the tumor cells was more frequent in the poorly differentiated carcinomas (44/51), but intestinal metaplasia was more pronounced in the adenocarcinomas (16/24). The poorly differentiated carcinomas were located in the basal region of the intestinal mucosa, and the glandular adenocarcinomas were located in the tegmental epithelium. (31 refs.)

\* (Review): 77-1835, 77-1836, 77-1837, 77-1838, 77-1839, 77-1840, 77-1843, 77-1844.

\* (Chem): 77-1861, 77-1863, 77-1873, 77-1874, 77-1879, 77-1880, 77-1900, 77-1904, 77-1908, 77-1917, 77-1926.

\* (Phys): 77-1965, 77-1968, 77-1972, 77-1979, 77-1980, 77-1981, 77-1984, 77-1997, 77-2000.

\* (Viral): 77-2027, 77-2046, 77-2048, 77-2053.

\* (Immun): 77-2085, 77-2172, 77-2178, 77-2180, 77-2182, 77-2183, 77-2186, 77-2187, 77-2188, 77-2189, 77-2191, 77-2192, 77-2193, 77-2232, 77-2233.

\* (Epid): 77-2287, 77-2288, 77-2294.

## EPIDEMIOLOGY AND BIOMETRY

- 77-2285 **Cancer Epidemiology. A Continuing Series on Different Sites, Based on the New South Wales Data.** (Eng.) Ford, J. (Central Cancer Registry, Health Commission New South Wales, CPO Box 4235, Sydney, New South Wales 2001, Australia) *Cancer Forum* (10): 348-350; 1977.

The number of new cases of melanoma in New South Wales in 1972 is reported along with the annual incidence/100,000 by age and sex, and subsite distribution by sex. Leading sites are tabulated by sex, showing percentage distribution, number of cases, and incidence rate. (no refs.)

- 77-2286 **Increased Incidence of Malignancy in Chronic Renal Failure.** (Eng.) Sutherland, G. A. (Renal Unit, St. Mary's Hosp., London, W2, England) Glass, J.; Gabriel, R. *Nephron* 18(3): 182-184; 1977.

The incidence of malignancy was examined in 120 patients seen for chronic renal failure (defined as having constant plasma creatinine levels of  $\geq 2.5$  mg/100 ml). No patient had received a kidney transplant or was therapeutically immunosuppressed for other reasons. Cancers were found in four patients, three of whom were on dialysis at the time of diagnosis. The mean time from the onset of uremia to the development of the cancer was 32.5 mo. No importance could be attached to age, sex, duration of uremia, or dialysis in relation to the four patients who developed neoplasms. However, the data do support the likelihood that a patient with chronic renal failure is more prone to develop cancer than a patient without renal disease. (7 refs.)

- 77-2287 **Comparative Histologic Study of Adenomas of the Large Intestine in Japan and England, with Special Reference to Malignant Potential.** (Eng.) Muto, T. (Dept. Surgery, Univ. Tokyo, Tokyo, Japan) Ishikawa, K.; Kino, I.; Nakamura, K.; Sugano, H.; Bussey, H. J.; Morson, B. C. *Dis Colon Rectum* 20(1): 11-16; 1977.

Colon tumors with at least some benign tissue were studied microscopically to compare the histologies of these tumors removed in Japan with those removed in England. Of the 2,506 tumors in England 59.4% were  $< 1$  cm diameter and 17.3% were  $> 2$  cm; 80.8% of 299 tumors in the Japanese material were  $< 1$  cm; and 4.3% were  $> 2$  cm. In England, both tubular and tubulovillous adenomas had greater malignant potential than those in Japan; the greater malignant potential of tumors in England was attribut-

able to their greater size. Villous adenomas were found in 1.3% of the tumors in Japan and in 9.7% of the tumors in England. These findings suggest that geographic factors influence the malignant potential of adenomas of the colon. (11 refs.)

- 77-2288 **Occurrence of Childhood Cancers Among Sibs and Estimation of Familial Risks.** (Eng.) Draper, G. J. (Childhood Cancer Res. Group, Old Radcliffe Observatory, Woodstock Road, Oxford, England) Heaf, M. M.; Wilson, L. M. *J Med Genet* 14(2): 81-90; 1977.

Cases of childhood cancer occurring in Britain over a 20-yr period were analyzed. It is suggested that there is a small familial element in the etiology of these diseases, as aggregations among sibs occurred more frequently than would be expected by chance. The cases among sibs may be due to associations between malignant disease and various genetically determined conditions at a subclinical level or in the heterozygous state. It is also possible that familial aggregations may be attributable to an environmental factor to which the sibs are exposed. Retinoblastoma families and sibs with leukemia, lymphoma, brain tumors, and adrenocortical carcinoma are discussed. The implications of the findings for genetic counselling are discussed. The main conclusion drawn was that if a child is diagnosed as having any of the aforementioned neoplasms, there is a small increase in the risk that a sib will also develop one of these diseases, and that it is likely that the two diagnoses will be concordant. (27 refs.)

- 77-2289 **Eye Color in Skin Cancer.** (Eng.) Prieto, J. G. (Martinez Campos 7, Madrid 10, Spain) *Int J Dermatol* 16(5): 406-407; 1977.

A study of 425 patients with skin cancer indicated that 359 (84.6%) of them had light-colored eyes. Only 15.4% of 1,750 patients with other skin conditions had light-colored eyes. (2 refs.)

- 77-2290 **Tumour Viruses Infect Us All.** (Eng.) Anonymous (No affiliation given) *New Sci* 74(1054): 526; 1977.

Recent studies have shown that most normal healthy people are probably infected with C-type tumor viruses, which contain RNA. In test animals these viruses make DNA copies



of their RNA genes which they then insert into the DNA of the host animal. This viral DNA can cause a cell to become cancerous in animals, but there is no evidence for this occurrence in humans. The isolation of C-type viruses from humans may potentiate the development of vaccines. (no refs.)

A 5-yr prospective follow-up covering women aged over 45 yr in the Uppsala region, Sweden, is planned for April 1977, to study the incidence of endometrial cancer among postmenopausal women using estrogen preparations vs those not using estrogens. (6 refs.)

- 77-2291 **Cervix Cancer in the Stockholm Region: Influence of Early Diagnosis on Morbidity and Mortality.** (Eng.) Einhorn, N. In: *Health Control in Detection of Cancer. Proceedings of a Symposium on Health Control in Detection of Cancer held by the Skandia Group, 23-25 September, 1975.* The Skandia Group. (Stockholm, Sweden): pp. 267-275; 1976.

This study analyzed the impact of early diagnosis of cervical cancer on the morbidity and mortality of the patients in the Stockholm area from 1951 to 1969. A total of 7,281 patients were involved in the study, and the number treated per year varied between 290 and 436. The relative incidence of cases in clinical Stage I increased from 12% in 1951 to 37% in 1969. The incidence of cases in Stage II decreased during the same period from 57% to 43%, and cases in Stage III decreased from 27% to 13%. The 5-yr survival rate for all patients was 57% from 1951 to 1953 and 59% from 1967 to 1969. The survival rate did not change despite earlier diagnosis due to the continuing loss of patients in Stage III of the disease. It is hypothesized that there are two biologically different types of cervical carcinoma: a less malignant type, which occurs with all precancerous stages and is easy to discover, and a more malignant type, which develops more quickly and is less liable to be discovered at an early stage. (17 refs.)

- 77-2292 **More Clues About the Pill and Liver Tumor Link.** (Eng.) Anonymous (No affiliation given) *JAMA* 237(25): 2701; 1977.

Reports of a possible connection between oral contraceptives and benign liver tumors were confirmed by a study conducted by the American College of Surgeons' Commission on Cancer. Of 378 women treated for benign tumors at 477 hospitals between 1970-1975, nearly 49% used birth control pills. Two benign tumors were most often associated with the contraceptives: liver cell adenomas and areas of focal nodular hyperplasia. (1 ref.)

- 77-2293 **Endometrial Cancer and Estrogen Treatment--An Epidemiological Study.** (Swe.) Liljestrand, A. (Socialstyrelsens lakemedelsavdelning, Sweden) Manell, P.; Westerholm, B.; Magnusson, B.; Stenson, S.; Persson, I.; Lindberg, B.; Johansson, E. *Lakartidningen* 74(13): 1258; 1977.

- 77-2294 **The Prevalence of Cervicitis, Reserve Cell Hyperplasia, Squamous Metaplasia, and Cervical Dysplasia in Jewish Women.** (Eng.) Czernobilsky, B. (Dept. Pathology, Kaplan Hosp., Rehovot, Israel) Zeituni, M.; Lancet, M.; Mazor, B.; Baram, A.; Deligdish, L. *Obstet Gynecol* 49(5): 587-591; 1977.

The prevalence of cervicitis, reserve cell hyperplasia, squamous metaplasia, and dysplasia was studied in Jewish women and compared with the statistics for non-Jewish populations. A histologic review was made of 250 women whose cervixes were either normal or undergoing benign changes and 50 women with squamous cell carcinoma. A heavy inflammatory infiltrate was present in 54.5% of those with benign conditions, but 78% of those with cervical carcinoma showed severe cervicitis. Reserve cell hyperplasia was noted in 22.4% of those with benign conditions and 8% of those with carcinoma. Squamous metaplasia was noted in 40.8% of those with benign conditions and 56% of those with carcinoma. Dysplasia was noted in 11 cases among those with benign conditions and in 30 of the carcinoma group. The prevalence of the histologic features in the two groups of Jewish women was found to be similar to that noted in comparable groups of non-Jewish women. (17 refs.)

- 77-2295 **The Incidence of Breast and Cervix Cancer in the Swedish Population.** (Eng.) Ericson, J. L.; Karnstrom, L.; Mattsson, B.; Willgren, J. In: *Health Control in the Detection of Cancer. Skandia International Symposium, September 23-25, 1975.* Bostrom, H.; Larsson, T., eds. (Stockholm: Almqvist & Wiksell International): pp. 191-204; 1976.

The incidence rates of breast and cervical cancer in Sweden during 1958-1971 were analyzed. From 1959-1965, the age-specific incidence of breast cancer increased rapidly and nearly exponentially from about age 30 (incidence rate 10/100,000) up to age 45 (140/100,000), followed by a slower rise that was also roughly exponential. From 1959 to 1971, the annual increase in breast cancer was 1.6%. The age-specific incidence of cervical cancer increased rapidly between ages 20 (1/100,000) and 44 (50/100,000), with a peak in the 40-44 age group. In both types of cancer there was a slight urban predominance. The increased incidence is felt to be partly due to better and earlier screening techniques. However, a different and more malignant type of disease is suspected of accounting for at least part of the increase. (no refs.)

**77-2296 Screening for Breast Cancer in Younger Women: Life Expectancy Gains and Losses. An Analysis According to Risk Indicator Groups.** (Eng.) Seidman, H. (Dept. Epidemiology and Statistics, Dept. Res., American Cancer Society, New York, NY) *CA* 27(2): 66-87; 1977.

Using data from Breast Cancer Detection Demonstration Projects (BCDDP) and from the American Cancer Society screening program of the 1960's, the risk-to-benefit ratio for screening younger women was calculated. Women with a breast lump or discharge or a history of breast surgery or breast cancer composed the major risk group. They showed breast cancer incidence rates > 100% in excess of the women with no major risk indicators. Women with menarche prior to age 12 or who had not given birth to a live child before the age of 30 yr were included in the minor risk group, which showed an excess of 15% to 50% over the breast cancer incidence rates of women with no risk factors. The usual clinical invasive breast cancer patient is subject to a loss of 21 yr of life at ages 35-39 yr and about half that at ages 55-59 yr, compared with normal life expectancy. The gain in life expectancy through screening was determined to be approx 5-7 yr for those < 50 yr and 2-4 yr for those in their 50's. The estimated detection benefit is many times the presumed radiation loss at initial screening, except in 35- to 39-yr-old women. The expectation that the greater frequency of breast cancer in high-risk women would yield an increased gain in life expectancy through screening is validated by the data. (25 refs.)

**77-2297 Characteristics of Breast Cancer in Younger and Older Women.** (Jpn.) Miura, S. (Aichi Cancer Center Hosp., Aichi, Japan) Yoshida, M.; Murai, H. *Jpn J Cancer Clin* 23(3): 177-185; 1977.

From 1964 to 1974 1,100 patients were treated surgically for breast cancer at the Aichi Cancer Center Hospital. Age-distribution analysis showed two peaks, one at 45-50 yr and the other at 60-65 yr. The low incidence of breast cancer in Japan correlated with the comparatively small peak at 60-65 yr. The clinical features of breast cancer patients < 35 yr of age and those > 65 yr were studied. Patients < 35 yr had a poorer prognosis than the elderly patients. (16 refs.)

**77-2298 Is Breast Cancer Caused by Radiation? (Meeting Abstract).** (Eng.) Simon, N. (Mount Sinai Sch. Medicine, New York, NY 10029) Silverstone, S. M. *Int J Radiat Oncol Biol Phys* 2(Suppl 1): 91; 1977. (no refs.)

**77-2299 Breast Cancer Incidence Among Atomic Bomb Survivors, Hiroshima and Nagasaki, 1950-1969 (Meeting Abstract).** (Eng.) McGregor, D. H. (Radiation Ef-

fects Res. Foundation, Hiroshima, Japan) Lang, C. E.; Choi, K.; Tokuoka, S.; Liu, P. I.; Wakabayashi, T.; Beebe, G. W. *Radiat Res* 70(3): 670; 1977. (no refs.)

**77-2300 Radiation-Induced Neoplasia (Meeting Abstract).** (Eng.) Colman, M. (Radiation Oncology Dept., Michael Reese Hosp. and Medical Center, Chicago, IL 60616) Kirsch, M.; Creditor, M. *Radiat Res* 70(3): 670; 1977. (no refs.)

**77-2301 Occupational Exposures to Radiolumineous Dial Painters (Meeting Abstract).** (Eng.) Moghissi, A. A. (Georgia Inst. Technology, Atlanta, GA) Simpson, R. E. *Radiat Res* 70(3): 666; 1977. (no refs.)

**77-2302 Mortality of Workers Exposed to Chloroprene (Meeting Abstract).** (Eng.) Pell, S. (Medical Div., E.I. du Pont de Nemours and Co., Wilmington, DE) *J Occup Med* 19(4): 286-287; 1977. (no refs.)

**77-2303 Pleural Complications from Asbestos Exposure (Meeting Abstract).** (Eng.) Gaensler, E. A. (Thoracic Surgery, Univ. Hosp., Boston, MA) *J Occup Med* 19(4): 286; 1977. (no refs.)

**77-2304 Occupational Histology of Lung Cancer (Meeting Abstract).** (Eng.) Morton, W. E. (Environmental Medicine Div., Univ. Oregon Medical Sch., Portland, OR) Treyve, E. L.; Goldsmith, D. F. *J Occup Med* 19(4): 286; 1977. (no refs.)

**77-2305 "Tar" and Nicotine Content of Cigarette Smoke in Relation to Death Rates.** (Eng.) Hammond, E. C. (Dept. Epidemiology and Statistics, American Cancer Society, New York, NY 10017) Garfinkel, L.; Seidman, H.; Lew, E. A. *Environ Res* 12(3): 263-274; 1976.

Nicotine (N) and "tar" (T) content of cigarette smoke is assessed in relation to death rates. More than 1,000,000 women and men who enrolled in an epidemiological investigation in 1959-1960 were traced for 12 yr. The adjusted number of deaths was 4,735.5 for "high" T/N smokers, 4,299.9 for



"medium" T/N smokers, and 3,991.2 for "low" T/N smokers. The difference between the "high" group and the "low" group was statistically significant. The adjusted number of lung cancer deaths was 318.4 for "high" T/N smokers, 285.5 for "medium" T/N smokers, and 235.2 for "low" T/N smokers. The difference between the "high" T/N group and the "low" T/N group was statistically significant. The mortality ratios were calculated by dividing the adjusted number of deaths in the "medium" T/N and "low" T/N group by the corresponding adjusted number of deaths in the "high" T/N group. The mortality ratio for the "low" T/N group was 0.84 for all causes of death combined and 0.74 for lung cancer deaths. The lung cancer mortality ratios for "low" T/N smokers were lower for women than for men. Mortality ratios were also calculated by dividing the adjusted death rates of the "medium" T/N and "low" T/N smokers by the adjusted death rates of the "high" T/N smokers. These mortality ratios were close to the previous results. The adjusted number of coronary heart disease deaths was 1,616.8 in "high" T/N smokers, 1,483.3 in "medium" T/N smokers, and 1,392.7 in "low" T/N smokers. The difference between the "high" T/N group and the "low" T/N group was statistically significant. The death rates of individuals who never smoked regularly were significantly lower than the death rates of individuals who smoked "low" T/N cigarettes. The adjusted numbers of deaths (total deaths, lung cancer deaths, and coronary heart disease deaths) are consistently lower among "low" T/N smokers than among "high" T/N smokers when the subjects are matched on number of cigarettes smoked per day. (11 refs.)

- 77-2306 **Fluoridation of Water and Cancer Mortality in the U.S.A.** (Eng.) Doll, R. (Dept. Regius Professor Medicine, Radcliffe Infirmary, Oxford OX2 6HE, England) Kinlen, L. *Lancet* 1(8025): 1300-1302; 1977.

The association between water fluoridation and cancer mortality in the US was assessed. Between 1950 and 1970, the proportions of the population that were nonwhite and > 65 yr of age increased more rapidly in the fluoridated than in the nonfluoridated cities. For a condition such as cancer, which increases in incidence rapidly with age, it was essential to make comparison between narrow age groups. When account was taken of age, sex, and ethnic group, the ratio between the observed cancer mortality and expected cancer mortality did not change in the nonfluoridated cities, and it

fell slightly in cities with fluoridated water. This evidence, when analyzed in detail, is consistent with that found in Britain. None of it provides any reason to suppose that fluoridation is associated with an increase or cause of cancer mortality. (11 refs.)

- 77-2307 **The Multistage Theory of Carcinogenesis (Letter to Editor).** (Eng.) Moolgavkar, S. H. (Dept. Epidemiology and Public Health, Univ. Washington, Seattle, WA 98195) *Int J Cancer* 19(5): 730; 1977.

The use of the multistage theory of carcinogenesis for studying the age distribution of cancer in man is discussed. Due to the need for approximation and for fitting incidence curves to the data, the application of the theory to acute leukemia, Hodgkin's disease, and lung cancer may not provide valid epidemiological evidence. (3 refs.)

- 77-2308 **Drinking Water: Health Hazards Still Not Resolved.** (Eng.) Wade, N. (No affiliation given) *Science* 196(4297): 1421-1422; 1977.

Some of the organic chemicals present in drinking water are known to cause cancer in laboratory animals, but it is unknown whether the minute amounts in drinking water present a threat to human health. The quarrel between the Environmental Defense Fund and the Environmental Protection Agency over the enforcement of the Safe Drinking Water Act of 1974 is discussed. (no refs.)

- \* (Review): 77-1802, 77-1806, 77-1808, 77-1809, 77-1810, 77-1811, 77-1812, 77-1813, 77-1814, 77-1815, 77-1816, 77-1817, 77-1837, 77-1840, 77-1841, 77-1842, 77-1844, 77-1846, 77-1848.  
\* (Chem): 77-1866, 77-1886, 77-1891, 77-1908.  
\* (Phys): 77-1980, 77-2013.  
\* (Path): 77-2243, 77-2253, 77-2284.

## MISCELLANEOUS

- 77-2309 **Purine and Pyrimidine Ribonucleotide Contents of Rat Liver and Hepatoma 3924A and the Effect of Ischemia.** (Eng.) Jackson, R. C. (Lab. Experimental Oncology, Indiana Univ. Sch. Medicine, Indianapolis, IN 46202) Boritzki, T. J.; Morris, H. P.; Weber, G. *Life Sci* 19(10): 1531-1536; 1976.

Concentrations of 12 ribonucleotides during ischemia in normal rat liver and rapidly growing hepatoma 3924A were studied. The hepatoma was carried as an sc transplant in male ACI/N rats. Tissues were rapidly excised and freeze-clamped within 1-2 sec; for ischemia samples, tissues were excised and kept at 23 C for 10 min prior to freeze-clamping. Liquid nitrogen-cooled metal tongs were used for freeze-clamping. Guanosine monophosphate (GMP) levels in the tumor were 325% of the control liver value ( $p < 0.05$ ). The concentration of cytidine triphosphate (CTP) was  $> 50\times$  that of ATP in the liver; this ratio was  $< 5$ -fold in the hepatomas. During 10 min of ischemia, the ATP content of the liver fell by 76%, ADP remained constant and AMP increased; a decrease in the total content of adenine nucleotides was accompanied by a striking increase in inosine monophosphate. Liver CTP and GTP levels declined during ischemia. Very little change occurred in the nucleotide pools of the hepatoma during 10 min of ischemia, possibly because of its low adenylate kinase activity and rapid anaerobic glycolysis. (14 refs.)

- 77-2310 **Effects of Increased Intracellular  $\text{Ca}^{2+}$  on Cyclic Nucleotides Production by Liver Tissue.** (Eng.) Anghileri, L. J. (New England Radiopharmaceutical Div., Atomlight Place, North Billerica, MA 01862) Heidebreder, M. *Experientia* 33(4): 415-416; 1977.

In order to correlate the variations of intracellular liver calcium with the changes in rate of cyclic AMP and cyclic GMP synthesis, male Wistar rats were exposed to hepato-intoxication with thioacetamide (TAA). Results showed that an increase in intracellular  $\text{Ca}^{2+}$  was accompanied by an increase in cyclic AMP, while  $\text{Mg}^{2+}$  and cyclic GMP concentrations corresponded to those observed in normal liver tissue. A direct relationship between  $\text{Ca}^{2+}$  concentration and cyclic AMP synthesis was indicated. (10 refs.)

- 77-2311 **An Altered Response to Cyclic AMP Stimulating Hormones in Intact Human Leukemic Lymphocytes.** (Eng.) Polgar, P. (Dept. Biochemistry, Boston Univ. Medical Center, Boston, MA 02118) Vera, J. C.; Rutenburg, A. M. *Proc Soc Exp Biol Med* 154(4): 493-495; 1977.

Lymphocytes from normal subjects and patients with chronic

lymphatic leukemia (CLL) were assayed for cyclic AMP content and their response to effector stimulation in terms of increased cellular cyclic AMP levels. Each CLL cell contained approx twice the cyclic AMP of normal lymphocytes. Both isoproterenol (ISP) and epinephrine (EP) showed stimulation relative to the basal soln in normal cells, but glucagon (GLC) had no appreciable effect. The relative response to EP was significantly reduced in the malignant cells, but GLC again had no effect on cyclic AMP levels. This was best shown in the stimulated/basal ratio, which indicated that the CLL response to EP was reduced by a factor of at least 2. The response to ISP also appeared to be decreased. In normal lymphocytes, prostaglandins  $\text{E}_1$  and  $\text{E}_2$  demonstrated approx an eightfold stimulation over basal value. Prostaglandin  $\text{F}_{2\alpha}$  was the least active and showed a fivefold stimulation over basal value. The leukemic cells displayed a decreased response to all three prostaglandins to approx one-third to one-fourth of that in normal lymphocytes. Propranolol ( $5 \times 10^{-6}$  M) inhibited ISP and EP stimulation by 80% and 95%, respectively, but it did not affect cyclic AMP levels consequent to prostaglandin stimulation. A dysfunction in receptor-adenylate cyclase interaction may exist within the membrane of leukemic cells. (11 refs.)

- 77-2312 **Control of Adenosine Monophosphate Catabolism in Mouse Ascites Tumor Cells (Meeting Abstract).** (Eng.) Sauer, L. A. (The Mary Imogene Bassett Hosp., Cooperstown, NY 13326) *Fed Proc* 36(3): 691; 1977. (no refs.)

- 77-2313 **Correlation Between Changes in Intracellular Level of Cyclic AMP, Activation of Cyclic AMP-Dependent Protein Kinase and the Morphology of Chinese Hamster Ovary Cells in Culture (Meeting Abstract).** (Eng.) Li, A. P. (Univ. Tennessee Oak Ridge Graduate Sch. Biomedical Science, Oak Ridge, TN 37830) O'Neill, J. P.; Kawashima, K.; Hsie, A. W. *Fed Proc* 36(3): 688; 1977. (no refs.)

- 77-2314 **Possible Role of Branch Migration in Excision and Replication Repair (Meeting Abstract).** (Eng.) Strauss, B. (Univ. Chicago, Dept. Microbiology, Chicago, IL 60637) *Mutat Res* 46(2): 158-159; 1977. (no refs.)

- 77-2315 **Characterization of Foldback Sequences in Hamster DNA Using Electron Microscopy.** (Eng.) Bell, A. J. (Dept. Biochemistry, Univ. Aberdeen, Ma-



rischal Coll., Aberdeen AB9 1AS, Scotland) Hardman, N. *Nucleic Acids Res* 4(1): 247-268; 1977.

Electron microscopy was used to characterize unfractionated hamster foldback DNA and to examine foldback molecules bound to hydroxyapatite crystals. It was found that the average length of the inverted sequences in the foldback molecules was about 0.9 kilobases. It was estimated that there were about 42,000 such sequences in the hamster genome, of which approx 45% formed loop structures, with a mean loop length of 1.74 kilobases. The binding of renatured duplex molecules to hydroxyapatite resulted in a poor recovery of structures containing identifiable foldback sequences. The relative proportion of contaminating intermolecular duplexes increased about three- to fourfold in the hydroxyapatite-bound fraction. (27 refs.)

**77-2316 Induction of Human Chorionic Gonadotropin in Hela Cultures by Inhibitors of DNA Synthesis and Cell Division (Meeting Abstract).** (Eng.) Ghosh, N. K. (New York Univ. Medical Center, New York, NY 10016) Rukenstein, A.; Cox, R. P. *Fed Proc* 36(3): 910; 1977. (no refs.)

**77-2317 Methylation of Newly Synthesized Extracellular DNA in the Lymphocytes of Healthy Persons and Patients with Chronic Lymphatic Leukemia.** (Rus.) Fedorov, N. A. (Lab. Biochemistry, Central Inst. Hematology and Blood Transfusion, USSR Ministry Public Health, Moscow, USSR) Borovkova, T. V.; Sevastianova, M. G. *Probl Gematol Pereliv Krovi* 21(11): 17-21; 1976.

Extracts of the lymphocytes from 14 healthy donors and 12 patients with chronic lymphocytic leukemia (CLL) were examined for any nonscheduled DNA synthesis. Incorporation of <sup>3</sup>H-deoxycytidine into DNA was significantly higher in leukemic WBC. As reported previously, lymphocytes from the CLL patients contained deoxycytidine deaminase, but those of the donor lacked this enzyme. It was concluded that the observed difference between normal lymphocytes and leukemia cells is important in elucidating the biochemical specificity of leukemic transformation. (12 refs.)

**77-2318 DNA Synthesis in Unstimulated Blood Lymphocytes of Patients with Untreated Malignant Lymphomas or Other Malignant Tumors.** (Eng.) Heier, H. E. (Norwegian Radium Hosp. and Norsk Hydro's Inst. Cancer Res., Lab. Haematology and Lymphology, Oslo, Norway) Godal, T. *Scand J Haematol* 2(18): 149-153; 1977.

DNA synthesis was examined prior to therapy in the un-

stimulated peripheral blood lymphocytes from 18 patients with Hodgkin's disease, 11 with nonleukemic lymphosarcoma, 12 with reticulosarcoma, 20 with various solid tumors and from 37 normal donors. All patient groups had significantly increased values compared to the controls, but no significant differences were observed among the patient groups. Malignant lymphoma patients with general symptoms had the highest median value; however, the range of values was similar for patients with and without general symptoms, with no statistically significant difference between these groups ( $p > 0.10$ ). This trend appeared to be consistent for all three lymphoma groups, but the numbers of patients with general symptoms within each lymphoma group were too small to make statistically significant calculations. These results indicate that both normal subjects and patients with malignant disease have some mononuclear cells that synthesize DNA. (15 refs.)

**77-2319 DNA Synthesis in Polyamine Deficient Nuclei (Meeting Abstract).** (Eng.) Knutson, J. C. (Univ. Washington, Seattle, WA 98195) Morris, D. R. *Fed Proc* 36(3): 849; 1977. (no refs.)

**77-2320 Further Evidence in Support of Cell-Surface-associated Deoxyribonucleic Acid in Tumor Cells: An Autoradiographic Study.** (Eng.) Aggarwal, S. K. (Dept. Zoology, Michigan State Univ., East Lansing, MI 48824) *J Histochem Cytochem* 25(5): 359-370; 1977.

Autoradiographic studies using <sup>3</sup>H-thymidine were conducted as label to confirm the nature of platinum-pyrimidine complex binding to the cell surface. The ascites sarcoma-180 tumor system was used as a model. Light microscope examination of random autoradiograms from random thick sections of the tumor cells showed silver grains located predominantly over the nucleus. Some grains were also found in the cytoplasm and on the plasma membrane. These grains were absent in deoxyribonuclease-pretreated cells. A histogram based on 639 random cells showed 958 silver grains within 1.0 micron on either side of the plasma membrane. In both light and electron microscope autoradiograms, photopositive silver grain distribution peaks were observed over the nucleus and the plasma membrane. The results confirm the presence of cell-surface-associated DNA, but the source and function of this DNA remain unresolved. (55 refs.)

**77-2321 DNA Binding Site of Activated Glucocorticoid Receptor. Interaction with Pyridoxal-P (Meeting Abstract).** (Eng.) Litwack, G. (Fels Res. Inst. and Dept. Biochemistry, Temple Univ. Sch. Medicine, Philadelphia, PA) Cake, M. H. *Fed Proc* 36(3): 911; 1977. (no refs.)

**77-2322 Interaction of DNA and Liposomes as a Model for Membrane-mediated DNA Damage.** (Eng.)

Pietronigro, D. D. (Dept. Pathology, New York Univ. Medical Center, New York, NY 10016) Jones, W. B.; Kalty, K.; Demopoulos, H. B. *Nature* 267(5606): 78-79; 1977.

The damaging consequences to the biological activity of bacterial-transforming DNA in oxidizing liposomal suspensions are reported. Incubation mixtures of liposomes and *Bacillus subtilis* or calf thymus DNA were prepared. The concentration of DNA had no effect on the induction period, which was approx 21 hr. Lipid oxidation was retarded by the DNA in a concentration-dependent manner. This suggests that DNA interacts with the oxidizing model membrane system as a free-radical acceptor or a trapping agent. Methyl arachidonate-enriched phosphatidylcholine (PCMA) liposomes demonstrated a higher rate of oxidation than phosphatidylcholine (PC) liposomes. At various times during the experiment, DNA was isolated from both the PCMA and PC liposomal suspensions and assayed for transforming activity. At 16 hr the PCMA liposomes decreased the DNA transforming activity by a factor of 2, but the PC liposomes caused no damage. By 36 hr, however, the PCMA liposomes decreased the activity by a factor of 3,000, and PC liposomes decreased by a factor of 8. After 68 hr both suspensions showed a decrease in activity by a factor of 3,000. DNA incubated with PC and PCMA liposomes in standard saline citrate did not undergo autooxidation. The results indicate that oxidation of highly organized lipid bilayers can damage DNA. (30 refs.)

**77-2323 DNA Repair in *Bacillus subtilis*. II. Activation of the Inducible System in Competent Bacteria.** (Eng.)

Yasbin, R. E. (Dept. Microbiology, Pennsylvania State Univ., University Park, PA 16802) *Mol Gen Genet* 153(2): 219-225; 1977.

The role of the *Bacillus subtilis* 'SOS' system in DNA mediated transformation was investigated. The results indicated that the development of competence is correlated with the activation or derepression of the 'SOS' system. (48 refs.)

**77-2324 RNA Synthesis and Processing as a Measure of Phenotypic Variability in Cytodifferentiation and Neoplasia.** (Eng.)

Bynum, J. W. (Sch. Medicine, Southern Illinois Univ., Carbondale, IL 62901) Regan, J. D.; Volkin, E. *J Cell Physiol* 91: 1-14; 1977.

Cell lines of different differentiated states and proliferative rates were used to assess the interrelationship between cytodifferentiation and cell proliferation. The cell lines were RPMI-8226 (human myeloma), C1300 (mouse neuroblastoma), and HeLa HCAAT. The myeloma, neuroblastoma, and HeLa cells were pulse-labeled with <sup>3</sup>H-uridine and the RNA fractions isolated. No direct correlation between RNA

synthesis and cell proliferation rate was observed. HeLa and myeloma cells displayed steady-state labeling of RNA 3 hr after a chase period, but the RNA of neuroblastoma cells continued to incorporate the label after the same interval. The specific activity at 3 hr was three times greater in the HeLa and myeloma cells than in the neuroblastoma cells. It is unlikely that these differences between cell lines are attributable to the cell proliferation rate, because the doubling times between myeloma and HeLa and between myeloma and neuroblastoma differ by 7 and 9 hr, respectively. The chromatin-associated RNA turned over more rapidly in HeLa cells than in the other cell types. In all three types, approx 10% of the total labeled RNA was chromatin-associated. During morphological differentiation of neuroblastoma cells there is a two- to threefold increase in the stable chromatin-associated RNA. In HeLa cells, the increase was in rapidly turning over chromatin-associated RNA. In all three lines, the poly(A) RNA from the nonchromatin-associated RNA was found to be rapidly labeled and rapidly turned over. RNA fractions from the chromatin-associated RNA of HeLa and neuroblastoma cells exhibited the same characteristics. The myeloma cell RNA fraction from the chromatin-associated RNA was slowly labeled and relatively stable. The myeloma cells could not suppress specialized protein synthesis associated with differentiation, which was observed with the neuroblastoma cells. Ribosomal RNA precursor synthesis and processing appear to be related to phenotype more than genotype. Phenotypic variability, rather than cell proliferation rate, seems to be more directly related to the synthesis and distribution of RNA populations. (32 refs.)

**77-2325 Enzymic Synthesis of a DNA Copy of 5S RNA (Meeting Abstract).** (Eng.)

Ackerman, S. (Dept. Pathology, Univ. Pennsylvania Sch. Medicine, Philadelphia, PA 19174) Henner, D.; Furth, J. J. *Fed Proc* 36(3): 658; 1977. (no refs.)

**77-2326 The Relative Amounts of the Cytoplasmic RNA Species in Normal, Transformed and Senescent Cultured Cell Lines.** (Eng.)

Johnson, L. F. (Dept. Biochemistry, Ohio State Univ., Columbus, OH 43210) Abelson, H. T.; Penman, S.; Green, H. *J Cell Physiol* 90: 465-470; 1977.

The relative content of cytoplasmic RNA species was determined in a number of cultured cells by labeling them to equilibrium in medium containing <sup>32</sup>Po<sub>4</sub>. Mouse cell lines 3T3, 3T6, and 3T3 transformed with simian virus 40 (SV40) and with both SV40 and polyoma virus were used along with human cervical carcinoma cells (HeLa), hamster lung fibroblasts (V79), and human embryonic lung fibroblasts. The cells were labeled and then harvested, and the amount of label in each cytoplasmic RNA species was determined. The ratio of 28S to 18S ribosomal RNA (rRNA) did not vary significantly in the different cell types. There was little variation in the ratio of 4S and RNA to 18S rRNA except for the senescent



human diploid fibroblasts, which showed a 70% increase in this ratio. Significant variation was found in the different cell types in the ratio of poly(A) + mRNA (messenger RNA) to rRNA. The highest ratio (2.6%-2.7%) was found in cells with the shortest doubling times (3T6 and V79). The lowest ratio was observed in resting 3T3 cells and in the slowly growing senescent human diploid fibroblasts. Cells experiencing growth limitation due to either contact inhibition or senescence exhibit an mRNA to rRNA ratio about 30% lower than that observed in corresponding cells in the growing state. (20 refs.)

**77-2327 Post-Transcriptional Modification of RNA's of Normal and Cancer Cells (Meeting Abstract).** (Eng.) Singhal, R. P. (Dept. Chemistry, Wichita State Univ., Wichita, KS 67208) Delmez, B. L.; Hiesterman, G. W. *Fed Proc* 36(3): 660; 1977. (no refs.)

**77-2328 Determination of the Molecular Weights of mRNA Coding for Tyrosine Aminotransferase and Tryptophan Oxygenase from Rat Liver (Meeting Abstract).** (Eng.) Hofer, E. (Inst. für Zellforschung, Deutsches Krebsforschungszentrum, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany) Sekeris, C. E. *Hoppe Seylers Z Physiol Chem* 358(3): 251; 1977. (no refs.)

**77-2329 Detection of Globin Messenger RNA Sequences by Molecular Hybridization.** (Eng.) Ross, J. In: *Immune RNA in Neoplasia*. Fink, M. A., ed. (New York: Academic Press, Inc.) pp. 27-36; 1976.

Techniques for messenger RNA (mRNA) detection and quantitation using molecular hybridization are described. Nucleases were exploited that recognized only single-stranded molecules. RNA was incubated with <sup>3</sup>H-complementary DNA (cDNA) to form DNA-RNA hybrids. An assay was devised to detect radioactive globin mRNA. Excess unlabeled globin cDNA was incubated with radioactive cellular RNA. The reaction mixture was then treated with ribonucleases to hydrolyze nonannealed RNA sequences to acid-soluble nucleotides and oligonucleotides. Total cytoplasmic RNA was isolated from cells cultured in <sup>3</sup>H-uridine (200 µCi/ml) for 2 hr. When this RNA was incubated with all reactants except cDNA and then treated with ribonucleases A and T<sub>1</sub>, 0.08%-0.10% remained acid-precipitable. Based on specificity studies with the <sup>3</sup>H-cDNA probe and considering the purity of the globin mRNA utilized to make the cDNA, it appeared that the cDNA-dependent ribonuclease-resistant material was globin message. Globin cDNA can be used to detect radioactively labeled globin mRNA from mouse fetal liver cell cytoplasm. (26 refs.)

**77-2330 Cell-free Translation of Human Myeloma Cell Poly(A)-containing Messenger RNA (Meeting Abstract).** (Eng.) Yaffe, L. (Roche Inst. Molecular Biology, Nutley, NJ 07710) *Fed Proc* 36(3): 770; 1977. (no refs.)

**77-2331 Substitution of Guanine for a Specific Base in tRNA by Extracts of Ehrlich Ascites Tumor Cells.** (Eng.) Itoh, Y. H. (Dept. Molecular Biology, Sch. Medicine, Keio Univ., 35 Shinanomachi, Shinjuku-ku, Tokyo, Japan) Itoh, T.; Haruna, I.; Watanabe, I. *Nature* 267(5610): 467; 1977.

Purified *Escherichia coli* transfer tRNAs were tested as acceptors for guanylation by an enzyme isolated from Ehrlich ascites tumor. tRNA<sup>Tyr</sup>, tRNA<sup>Asp</sup>, tRNA<sup>Asn</sup>, and tRNA<sup>His</sup> were active as acceptors. The results obtained from chromatograms and acceptor activities of tRNAs indicated that the enzyme catalyzed the reaction to exchange the Q base for guanine at the first position of anticodon. The difference in guanylation activity among these tRNAs may be due to a modification of a nucleoside such as Q, or a specific structure or sequence may be recognized by the enzyme. (10 refs.)

**77-2332 Sequence Analysis of T1 Ribonuclease Fragments of 18S Ribosomal RNA by 5'-Terminal Labeling, Partial Digestion, and Homochromatography Fingerprinting.** (Eng.) Fuke, M. (Dept. Pharmacology, Baylor Coll. Medicine, Houston, TX 77030) Busch, H. *Nucleic Acids Res* 4(2): 339-352; 1977.

A method for sequence analysis of oligoribonucleotides that employs 5'-terminal labeling, endonucleolytic digestion, and homochromatographic fingerprinting is described. The sequence of ribonuclease T1 fragment 2 of 18S ribosomal RNA was determined to illustrate the application of this method. The results obtained with this sequencing technique were consistent with previously reported findings. The method described here can also be applied to RNA which is difficult to label in vivo, such as eukaryotic messenger RNA or RNA from human tissues. (19 refs.)

**77-2333 The Distribution of Acid and Alkaline Ribonuclease Activities in Preneoplastic and Neoplastic Rat Livers.** (Eng.) Murthy, S. M. (Dept. Immunochimistry Res., Evanston Hosp., Evanston, IL) *J Histochem Cytochem* 25(2): 115-121; 1977.

Ribonuclease (RNase) activities revealed by the substrate film method were compared with reactions for alkaline and acid RNases obtained by lead precipitation in hepatomas and preneoplastic livers. RNA films exposed to sections of normal rat livers revealed significant RNase activity in the liver

parenchyma. The cytoplasm and the nuclei of hepatocytes gave positive reactions for alkaline and acid RNases by lead precipitation. The bile canaliculi and the connective tissue cells demonstrated weak or negligible acid RNase activity, but they gave positive reactions for alkaline RNase. No lead precipitation was obtained in liver sections incubated in media without a substrate, and only faint reactions were noted in the absence of exogenous phosphatase. Rats fed 4-dimethylaminoazobenzene developed nodules of parenchymal cells in the liver after 2-3 mo of feeding. Concomitant with the development of hyperplastic nodules, a focal loss of RNase activity occurred. The nodules were almost completely deficient in RNase activity when a RNA film was exposed to adjacent serial sections, but the surrounding parenchymal tissue showed appreciable activity. Comparable results were obtained by lead precipitation. A more prolonged feeding of rats with the carcinogen resulted in foci of parenchymal cells characterized by intense staining of cytoplasmic RNA with basic dyes. The hyperbasophilic foci and the tumors were inactive against RNA films except for necrotic and stromal tissues that often gave positive reactions. The demonstration of acid RNase activity in adjacent serial sections revealed low activity in both the hyperbasophilic focus and the tumor. The positive reactions in some areas were associated with necrotic tissues. RNase activity was noted in necrotic zones of tumors. The cells located at the periphery of the necrotic areas and showing signs of degeneration were especially active. Parallel analyses of acid and alkaline RNase activities indicated increases in both enzyme activities in the same necrotic zone of the tumor mass. There is a good correlation between the RNase activities revealed by both methods. (17 refs.)

**77-2334 Glucosamine Phosphate Synthase of Neoplastic and Regenerating Liver.** (Eng.) Tsuiki, S.; Miya-gi, T. In: *Control Mechanisms in Cancer*. Criss, W. E.; Ono, T.; Sabine, J. R., eds. (New York: Raven Press): Progress in Cancer Research and Therapy, vol. 1, pp. 441-450; 1976.

Studies of the molecular alterations of glucosamine phosphate synthase in regenerating liver are described. Isoelectric focusing patterns have shown that glucosamine phosphate synthase isolated from the brain of rats was the major enzyme component of an ascites hepatoma (AH-130) and embryos 12 to 14 days old. Glucosamine phosphate synthase from rat liver was also the major form in a Morris hepatoma (5123D) and in fetal liver 19 days old. Two other forms different from each other and from the brain and liver forms were also detected. Hepatocarcinogenesis caused profound molecular alterations in hepatic glucosamine phosphate synthase. These alterations were usually in the direction of dedifferentiation. The effect of partial hepatectomy on the molecular composition of hepatic glucosamine phosphate synthase was studied. This procedure stimulated an elevation of hepatic glucosamine phosphate synthase levels. The activity began to rise before DNA synthesis and reached a peak value 70% above the level observed in controls. An increase in enzyme activity

was also noted following laparotomy and after injection of a mixture of triiodothyronine, amino acids, glucagon, and heparin (TAGH), indicating that elevation of enzyme level may have little relation to the proliferative character of the regenerating liver. Partial hepatectomy also induced qualitative changes in hepatic glucosamine phosphate synthase. On a hydroxyapatite column, hepatic synthase usually emerged as a single peak, but enzyme from 48 hr regenerating liver was not homogeneous and required greater concentrations of KCl for elution. Isoelectric focusing revealed similar patterns for enzyme from Morris hepatoma and from liver at initial stages after partial hepatectomy or TAGH injection. (17 refs.)

**77-2335 Changes in the 2,3-Diphosphoglycerate and ATP Contents in the RBC of Leukemia Patients.** (Rus.) Lukanova, I. S. (Leningrad Inst. Hematology and Blood Transfusion, Leningrad, USSR) Blinov, M. N.; Abdulkadyrov, K. M. *Probl Gematol Pereliv Krovi* 21(11): 26-29; 1976.

ATP and 2,3-diphosphoglycerate (2,3-DPG) were assessed in the RBC and sera of patients with different forms of leukemia. The 2,3-DPG level was elevated in patients with acute leukemia (18.7  $\mu\text{M}$ ) and chronic myeloid leukemia (20.1  $\mu\text{M}$ ) and decreased in patients with chronic lymphoid leukemia (12.8  $\mu\text{M}$ ) compared to controls (14.4  $\mu\text{M}$ ). ATP content was significantly increased in patients with chronic myeloid leukemia (4.0  $\pm$  0.32  $\mu\text{M}$ ) and decreased in patients with chronic lymphoid leukemia (2.7  $\pm$  0.21  $\mu\text{M}$ ). Patients with acute leukemia had values (3.5  $\mu\text{M}$ ) close to controls (3.5  $\mu\text{M}$ ). The decrease of ATP and 2,3-DPG in the patients with chronic lymphoid leukemia might be due to the reduced glycolytic activity in the RBC. (12 refs.)

**77-2336 Identification of Rat Arylsulfatase (AS) A and B in Basophil Leukemia (RBL) Tumors and Mast Cells (MC): Capacity to Inactivate SRS-A (Meeting Abstract).** (Eng.) Wasserman, S. I. (Harvard Medical Sch. and Robert B. Brigham Hosp., Boston, MA 02120) Austen, K. F. *Fed Proc* 36(3): 1328; 1977. (no refs.)

**77-2337 Differences in Adenylate Cyclase Activities in Murine Normal Cells and Bladder Tumor Cells in Tissue Culture.** (Eng.) Droller, M. J. (Dept. Surgery, Div. Urology, Stanford Univ. Medical Center, Stanford, CA 94305) *Invest Urol* 14(3): 249-252; 1976.

The differences in adenylate cyclase activities in murine bladder tumor cells and normal cells in tissue culture are investigated. The initial inoculum did not seem to alter the character of the adenyl cyclase activity changes observed. At day 1, adenyl cyclase base line activity was 674 picomoles of cyclic



AMP per  $10^6$  cells per 10 min. Max base line levels were reached by day 2, and averaged 1,077 picomoles of cyclic AMP per  $10^6$  cells per 10 min. After confluency had been reached, at day 4, enzyme activity decreased to half-max levels. Adenyl cyclase activity remained at initial base line levels through 9 days of incubation. Stimulation of adenyl cyclase activity of prostaglandin  $E_1$  ( $PGE_1$ ;  $3 \times 10^{-6}$  M) led to levels of cyclic AMP that were 25-30% greater than base line levels. Sodium fluoride stimulated base line levels 8-10x at each day tested through confluency. The bladder tumor cells demonstrated different patterns. The cells appeared to begin reaching a plateau of growth at 6 days, at which point there were an av of  $4.25 \times 10^5$  cells per  $cm^2$ . Base line adenyl cyclase activity in the bladder tumor cells was only one-fourth to one-fifth that of the corresponding BALB/c mouse embryo fibroblasts at each day tested. The peak level of activity was noted at day 2.  $PGE_1$ , stimulated adenyl cyclase levels in the bladder tumor cells six- to eightfold. On day 1, stimulated levels were still less than those seen in the embryo fibroblasts. By day 2,  $PGE_1$ -stimulated levels in the bladder tumor cells had overtaken cyclase levels in the fibroblasts and were an av of 25%-30% greater. When the ratio of  $PGE_1$ -stimulated activity in tumor as compared with normal cells was compared with the ratio of unstimulated base line levels, the tumor cells demonstrated a relatively far greater adenyl cyclase activity than did the nonneoplastic cells. Sodium fluoride had a less marked stimulatory effect on the bladder tumor cells (six- to eightfold) when compared to the embryo fibroblasts (eight- to tenfold). In view of the differential biochemical effect of the PG on tumor vs normal cells, a potential applicability to in vivo systems might be considered. (26 refs.)

77-2338 Regulation of Adenylate Cyclase Activities in Normal Rat Kidney and Morris Kidney Tumors (Meeting Abstract). (Eng.) Pradhan, T. K. (Howard Univ. Coll. Medicine, Washington, DC 20060) *Fed Proc* 36(3): 685; 1977. (no refs.)

77-2339 Alkaline Phosphatase Isoenzyme in Intestinal Metaplasia of the Stomach. (Eng.) Miki, K. (First Dept. Internal Medicine, Faculty Medicine, Univ. Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo, Japan) Oda, T.; Suzuki, H.; Iino, S.; Niwa, H. *Clin Chim Acta* 76(1): 79-88; 1977.

The properties of intestinal metaplastic mucosa alkaline phosphatase (ALP) were investigated and compared with those of metaplastic gastric mucosa ALP and intestinal ALP. Fiberscopic examination of 82 patients yielded 166 biopsy specimens of gastrointestinal mucosa. Zymograms of serum ALP showed the presence of seven active bands. The ALP activity of the metaplastic gastric mucosa ranged from 0.01 to 0.89 K-A unit/ $\mu g$  protein (av 0.15). The activity increased with extent of intestinal metaplasia in the specimen. Duodenal and jejunal mucosa ALP showed the highest activities,

followed by ileal, metaplastic gastric, duodenal bulb, and colonic mucosa ALP. No ALP activity was detected in normal gastric mucosa or esophageal mucosa. Metaplastic gastric mucosa ALP yielded inhibition curves with L-phenylalanine and imidazole that were similar to those of purified intestinal ALP. The heat-stability curves for these two ALP's also appeared similar. Metaplastic gastric ALP had a  $K_m$  value of 0.3 mM. Sensitivity to neuraminidase, double-immunodiffusion techniques, zymograms, dose-response curves, and immunofluorescent staining all showed metaplastic gastric ALP to be enzymologically and immunologically identical to purified human intestinal ALP. (25 refs.)

77-2340 Isozyme Characterization of Cultured Human Tumor Cell Lines (Meeting Abstract). (Eng.) Wright, W. C. (Sloan-Kettering Inst. for Cancer Res., Rye, NY 10580) Fogh, J. *Fed Proc* 36(3): 1079; 1977. (no refs.)

77-2341 Ultrastructural Localization of the Enzymes of Nuclear Membranes in Normal and Tumour Cells (Meeting Abstract). (Eng.) Raikhlin, N. T. (Inst. Experimental and Clinical Oncology, Acad. Medical Sciences USSR, Moscow, USSR) *Folia Histochem Cytochem (Krakow)* 14(4): 361; 1976. (no refs.)

77-2342 Malignant Schwannoma Growth and Ornithine Decarboxylase. (Eng.) Helson, L. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021) Rubenstein, R. *IRCS Med Sci: Cancer* 5(5): 229; 1977.

Human schwannoma, heterotransplanted and in vitro, was tested for the presence of an endogenous ornithine decarboxylase inhibitor following induction and exposure to putrescine. The inhibition of ornithine decarboxylase by putrescine occurred only during enzyme induction. The (3 refs.)

77-2343 Studies on the Multiple Molecular Forms of Cytosolic NAD-linked Glycerol-3-Phosphate Dehydrogenase from Normal and Neoplastic Rabbit Tissues (Meeting Abstract). (Eng.) Ostro, M. (Syracuse Univ., Syracuse, New York, NY 13210) Kornbluth, R.; Fondy, T. *Fed Proc* 36(3): 739; 1977. (no refs.)

77-2344 Inhibition of Tumor Collagenases by a Cartilage-Derived Inhibitor (Meeting Abstract). (Eng.) Kuettner, K. E. (Dept. Biochemistry, Rush Medical Coll., Chicago, IL 60612) Soble, L.; Croxen, R.; Hiti, J.; Harper, E.; Pauli, B. *Fed Proc* 36(3): 677; 1977. (no refs.)

77-2345 **Purification and Characterization of Rat Tumor Collagenase (Meeting Abstract).** (Eng.) Huang, C. C. (Dept. Otolaryngology, Univ. Iowa, Iowa City, IA 52242) Abramson, M. *Fed Proc* 36(3): 678; 1977. (no refs.)

77-2346 **Isolation and Partial Characterization of Plasma Membrane Protease from Adenocarcinoma of Human Pancreas (Meeting Abstract).** (Eng.) Harvey, S. R. (Roswell Park Memorial Inst., Buffalo, New York, NY 14263) Van Dusen, L. R.; Holyoke, E. D.; Chu, T. M. *Fed Proc* 36(3): 763; 1977. (no refs.)

77-2347 **Involvement of Protein Kinase in the Induction of Tyrosine Aminotransferase by cAMP Analogs (Meeting Abstract).** (Eng.) Wimalasena, J. (Dept. Pharmacology, Univ. Colorado Medical Center, Denver, CO 80262) Leichtling, B. H.; Su, M. Y.; Wicks, W. D. *Fed Proc* 36(3): 737; 1977. (no refs.)

77-2348 **Activation of Melanoma Tyrosinase by a Cyclic AMP-dependent Protein Kinase in a Cell-free System.** (Eng.) Korner, A. (Dept. Biology, Yale Univ., New Haven, CT 06511) Pawelek, J. *Nature* 267(5610): 444-447; 1977.

Melanin is synthesized in melanosomes starting with the oxidation of tyrosine to dihydroxyphenylalanine by the enzyme tyrosinase (T-ase), and experiments were conducted to identify the molecules controlling the activation of T-ase. A cell-free system was used in which T-ase is activated after the addition of a cyclic AMP (cAMP)-dependent protein kinase isolated from melanoma cells. An S30 fraction of PS1-HGPRT-1 cells (which have low basal T-ase activity) was incubated with or without cyclic cAMP-dependent protein kinase, cAMP, ATP, and Mg. T-ase activity increased dramatically after a lag of several hours. The activation kinetics were similar to those observed in intact cells. The activation was completely dependent on the protein kinase and was enhanced by cAMP, ATP, and Mg. It involved removal of an inhibitor of T-ase, as freshly thawed S30 fractions contained T-ase inhibitory activity that disappeared after T-ase activation. Phosphoprotein phosphatase activity was also present; it was shown to be distinct from the inhibitor, but it appeared to antagonize the kinase-mediated reaction. The lag in T-ase activation may be due to an antagonism between kinase-mediated phosphorylations and phosphatase-mediated dephosphorylations. It is concluded that these events are the same as those that occur in cultured cells. (10 refs.)

77-2349 **Solubilization and Characterization of Erythrocyte Membrane Protein Kinases (Meeting Abstract).** (Eng.) Hosey, M. M. (Univ. Illinois Medical Center, Chicago, IL 60612) *Fed Proc* 36(3): 641; 1977. (no refs.)

77-2350 **Poly (ADP-Ribose) Polymerase: The Distribution of a Chromosome-associated Enzyme Within the Chromatin Substructure (Meeting Abstract).** (Eng.) Mullins, D. W. (Dept. Biochemistry, Georgetown Univ., Washington, DC 20007) Giri, C. P.; Smulson, M. *Fed Proc* 36(3): 661; 1977. (no refs.)

77-2351 **Terminal Deoxynucleotidyl Transferase (TdT) Activity in Human Leukemic Cells and in Two Unique Cell Lines Derived from Them (Meeting Abstract).** (Eng.) Sahai Srivastava, B. I. (Dept. Experimental Therapeutics, Roswell Park Memorial Inst., Buffalo, New York, NY 14263) Minowada, J. *Fed Proc* 36(3): 735; 1977. (no refs.)

77-2352 **Stimulation of Microsomal Methyl Sterol Demethylase by Cytosolic Protein from Morris Hepatoma 7777.** (Eng.) Longino, M. A. (Section Biochemistry, Molecular & Cellular Biology, Cornell Univ., Ithaca, NY 14853) Gaylor, J. L.; Morris, H. P. *Fed Proc* 36(3): 779; 1977. (no refs.)

77-2353 **Cyclic Nucleotide Phosphodiesterase and Protein Activator in Human Cancer Cell Lines and Brown-Pierce Carcinoma (Meeting Abstract).** (Eng.) Liu, Y. P. (NIH, Bethesda, MD 20014) Wong, V.; Chabner, B. A. *Fed Proc* 36(3): 687; 1977. (no refs.)

77-2354 **Fibrinolysis Associated with Human Neoplasia: Production of Plasminogen Activator by Human Tumours.** (Eng.) Nagy, B. (Central Inst. for Tumours and Allied Diseases, Zagreb, Yugoslavia) Ban, J.; Brdar, B. *Int J Cancer* 19(5): 614-620; 1977.

The plasminogen-dependent fibrinolytic activity of several human tumor (cervical, mammary, prostatic, ovarian, lung carcinomas, melanoma, basalioma) was studied. Neoplastic tissues were obtained from cancer patients by either surgery or biopsies. Fibrinolysis in lysates either of tumor tissue specimens or of respective cell cultures was measured based on release of radioactivity from  $^{125}\text{I}$ -fibrin-coated Petri dishes. All examined tumors showed elevated levels of plasminogen-dependent fibrinolytic activity. However, normal control tis-



sues had little or no activity. These results indicate that fibrinolysis might be useful in differentiating between malignant and normal tissues. (20 refs.)

- 77-2355 Production of Plasminogen Activator by Established Cell Lines of Mouse Origin.** (Eng.) Rifkin, D. B. (The Rockefeller Univ., New York, NY 10021) Pollack, R. *J Cell Biol* 73(1): 47-55; 1977.

Tests on 17 established mouse cell lines and their transformants showed that the correlation between the production of plasminogen activator (PA) and neoplastic transformation is not always conserved. In cell lines derived from BALB/c 3T3 most of the transformed lines examined synthesized no more PA than the parental nontransformed BALB/c 3T3 A31CL10 cells. Cell line KA31 synthesized low levels of PA but was highly tumorigenic, showing that increased PA synthesis is not necessary for tumor formation. Swiss 3T3 transformant SV101 showed a higher level of PA than the parental cell. Two phenotypic partial revertants also produced large amounts of PA. Two cell lines (Aγ7 and SV101) with high production of PA produced tumors when injected into nude mice, while two cell lines (3T3 [Col 2] and F1SVII) that produced little PA were not tumorigenic. (22 refs.)

- 77-2356 Release of Fibrinolytic Activators from Human Ovarian Tumors in Organ Culture.** (Eng.) Svanberg, L. (Dept. Obstetrics and Gynaecology, Univ. Lund, Malmö Allmänna Sjukhus, S-214 01 Malmö, Sweden) *Ann Chir Gynaecol Fenn* 65(6): 405-407; 1976.

The release of fibrinolytic activators from human ovarian tumors was investigated. Tissue specimens were cultured in the presence of, but not in direct contact with, a standard fibrin clot. The fibrinolytic activators released from the cells passed into the medium, reached the clot by diffusion, converted its contaminating plasminogen into plasmin, and caused its gradual breakdown into fibrin/fibrinogen degradation products (FDP). Determination of the FDP provided an indirect measure of the amount of the fibrinolytic activators released. The material consisted of different ovarian tumors from 27 patients and normal ovaries from seven. From each tumor or ovary, four cultures were prepared. The medium was assayed every 24 hr. At the end of the culture period (3 days), the explants were examined histochemically for fibrinolytic activators. In addition, ascitic cells from three serous cystadenocarcinomas were washed in saline and incubated in the same test system. FDP appeared in progressively increasing amounts in all media. The tumors varied considerably in fibrinolytic activity, with the highest occurring in the mesonephroma. High fibrinolytic activity was also observed in the ascitic cells. The addition of tranexamic acid, an inhibitor of fibrinogen activation, completely inhibited clot degradation. Routine histological examination to exclude any toxic effect of tranexamic acid revealed good survival of the explants.

FDP did not appear in the medium from the controls. (12 refs.)

- 77-2357 Cell Surface Fibrinogen/Fibrin (FIB) Receptors on Cultured Human Fibroblasts: Relationship to Cold-Insoluble Globulin and Viral Transformation (Meeting Abstract).** (Eng.) Colvin, R. B. (Dept. Pathology, Massachusetts General Hosp., Boston, MA 02114) Gardner, P. I.; Roblin, R. O.; Verderber, E. L.; Mosesson, M. W. *Fed Proc* 36(3): 316; 1977. (no refs.)

- 77-2358 Molecular Composition and Origin of Substrate-attached Material from Normal and Virus-transformed Cells.** (Eng.) Culp, L. A. (Dept. Microbiology, Sch. Medicine, Case Western Reserve Univ., Cleveland, OH 44106) *J Supramol Struct* 5(2): 239-255; 1977.

Cellular components that adhere to the tissue culture substrate after ethylenediamine(oxyethylenetriamino)tetraacetic acid (EGTA)-mediated removal of normal BALB/c 3T3, simian virus 40 (SV40)-transformed 3T3, and concanavalin A-selected revertant cells have been characterized. Analyses indicated that the substrate-attached material (SAM) contains three size classes of hyaluronate proteoglycans, a large-external-transformation-sensitive (LETS) glycoprotein, a myosinlike protein (actin), and a few unidentified proteins. The LETS glycoprotein and one of the unidentified proteins appear in greater relative quantities in newly synthesized SAM than in the SAM accumulated over a long period of time. These two components also turn over very rapidly following short radio-labeling periods during chase analysis. The SAM's deposited during a wide variety of cellular attachment and growth conditions contained the same components in similar relative proportions, which may indicate well-controlled and coordinate deposition of a cell-surface complex involving the hyaluronate proteoglycans, the LETS glycoprotein, actin-containing microfilaments with associated proteins, and a number of other proteins in the substrate adhesion site. Virus-transformed cells deposited less SAM protein and polysaccharide quantitatively, although the qualitative composition of the SAM's and their metabolic behavior were similar. Much more evidence is required to determine if subtle qualitative differences in these SAM's exist. (39 refs.)

- 77-2359 An Attempt to Determine the Composition of Surface Glycoproteins in the Cells of Solid Melanomas in the Golden Hamster (Meeting Abstract).** (Eng.) Kozłowska, K. (Dept. Histology, Inst. Medical Biology, Medical Acad., Gdansk, Poland) Bomirski, A.; Zurawska-Czupa, B. *Folia Histochem Cytochem (Krakow)* 14(4): 345; 1976. (no refs.)

**77-2360 Surface Protein Components of Hepatoma Tissue Culture Cell Plasma Membranes (Meeting Abstract).** (Eng.) Levy, D. (Dept. Biochemistry, Univ. Southern California Sch. Medicine, Los Angeles, CA 90033) McQueen, H. M.; Cheng, S. *Fed Proc* 36(3): 703; 1977. (no refs.)

**77-2361 Cell Surface Protein and Neoplastic Transformation.** (Eng.) Yamada, K. M. (Lab. Molecular Biology, NCI, NIH, Bethesda, MD 20014) Pastan, I. *Trends Biochem Sci* 1(4): 222-224; 1976.

The properties of the major cell surface protein (CSP) and experiments that suggest its role in malignant transformation are reviewed. CSP is a major cell protein, and it is the major cell membrane protein of chick embryo fibroblasts. CSP is heavily labeled by techniques that only label proteins present on the outside of cells. It is diminished or absent after transformation by oncogenic viruses, but in confluent nongrowing cells it is present in increased amounts. CSP is rapidly destroyed by proteolytic enzymes. Reconstitution experiments suggest that diminished CSP content is partly responsible for the abnormal adhesiveness, shape, and movement of transformed fibroblastic cells. (29 refs.)

**77-2362 Rat Liver Mitochondrial Protein Synthesis In Vivo after Administration of Cycloheximide (CHI) (Meeting Abstract).** (Eng.) Froman, P. A. (Hahnemann Medical Coll. Hosp., Philadelphia, PA 19102) Devlin, T. M.; Ch'ih, J. J. *Fed Proc* 36(3): 647; 1977. (no refs.)

**77-2363 Effect of Amino Acid Starvation and Addition of Cycloheximide on the Rate of  $^3\text{H}$ -Uridine Incorporation into Yeast RNA Species (Meeting Abstract).** (Eng.) Kelker, H. C. (The Lindsley F. Kimball Res. Inst. The New York Blood Center, New York, NY 10021) Gross, K. J.; Pogo, A. O. *Fed Proc* 36(3): 659; 1977. (no refs.)

**77-2364 The Effect of Dexamethasone on the Non-histone Chromosomal Proteins of Hepatoma Tissue Culture Cells.** (Eng.) Edwards, R. J. (Dept. Biochemistry, Univ. Glasgow, Glasgow G12 8QQ, Scotland, United Kingdom) Knowler, J. T. *Biochem Soc Trans* 4(6): 1114-1116; 1976.

The influence of dexamethasone on the nonhistone chromosomal proteins of hepatoma tissue culture cells was investigated. Hepatoma tissue culture cells were grown in Dulbecco's medium supplemented with 10% calf serum and 2% fetal calf serum. The serum was treated with charcoal to remove endogenous corticosteroid. Dexamethasone was added to the medium at 5  $\mu\text{M}$ . There was neither gross stimu-

lation of nonhistone chromosomal protein turnover nor phosphorylation due to dexamethasone. Furthermore, when protein preparations were fractionated by one-dimensional polyacrylamide gel electrophoresis, no differences were detected in stainable bands or labeling with  $^3\text{H}$ -tryptophan. However, when the proteins were fractionated by a two-dimensional system, utilizing isoelectric focusing and sodium dodecyl sulfate/urea/polyacrylamide gel electrophoresis, a small hormone-dependent difference in the stable-protein pattern was detected. When the nonhistone chromosomal proteins were labeled with [ $^{32}\text{P}$ ]Pi and care was taken to digest all the nucleic acids, a small but reproducible change was detected in the labeling profile by one-dimensional polyacrylamide gels. A peak of phosphate incorporation was decreased within 1 hr of hormone treatment. Thus, a corticosteroid-induced change in the nonhistone chromosomal protein of hepatoma tissue culture cells was demonstrated. (9 refs.)

**77-2365 Ultrastructural Cytochemistry of Active Chromatin in Regenerating Rat Hepatocytes.** (Eng.) Derenzini, M. (Istituto di Patologia Generale dell'Universita, Via S. Giacomo 14, I-40126 Bologna, Italy) Lorenzoni, E.; Marinozzi, V.; Barsotti, P. *J Ultrastruct Res* 59(3): 250-262; 1977.

Ultrastructural alterations in the regenerating hepatocytes of Wistar rats were studied 24 hr after partial hepatectomy and 4 hr after injection of  $\alpha$ -amanitin (50  $\mu\text{g}/100\text{ g}$ , ip). Double staining of liver specimens with uranyl acetate and lead citrate revealed a marked increase of loosened electron-dense material, ie, decondensed chromatin, in hepatocyte nuclei 24 hr after hepatectomy. The Bernhard regressive EDTA staining method showed that the material was composed mainly of perichromatin fibrils. The Gautier osmium-ammine staining for DNA demonstrated that, at this time of regeneration, the DNA-containing structures were composed of thin threads that contributed, although in a small quantity, to the formation of the electron-dense material.  $\alpha$ -Amanitin induced a marked condensation of the loosened material: the quantity of perichromatin fibrils was strongly reduced, but the DNA-containing structures were condensed in compact masses. The perichromatin fibrils were detected only in areas where the DNA-containing structures were in the form of thin threads. The quantity of perichromatin fibrils was almost proportional to the quantity of thin DNA fibers. (26 refs.)

**77-2366 Isolation of Human Leukokininogen from Ascites of Ovarian Carcinoma (Meeting Abstract).** (Eng.) Roffman, S. (Columbia Univ., Coll. Physicians and Surgeons, New York, NY 10032) Greenbaum, L. M. *Pharmacologist* 18(2): 230; 1976. (no refs.)



- 77-2367 Distribution of Lectin Binding Sites in Various Subcellular Fractions of Raji Lymphoid Cells (Meeting Abstract).** (Eng.) Jett, M. (Blood Res. Lab., American Natl. Red Cross, Bethesda, MD 20014) *Fed Proc* 36(3): 711; 1977. (no refs.)
- 77-2368 Ratio of Membrane-bound and Free Ribosomes in the Livers of Healthy Rats, Rats with Zajdela's Ascites Hepatoma, and in the Ascites Cells Themselves.** (Eng.) Pushkina, I. P. (Lab. Tumor Biochemistry, Oncological Scientific Center, Acad. Medical Sciences USSR, Moscow, USSR) Kretchetova, G. D.; Shapot, V. S. *Biokhimiya (NY)* 41(11): 1573-1577; 1977.
- Standard fractionation conditions were used to isolate membrane-bound and free ribosomes in the livers of healthy rats, in the livers of rats with developing Zajdela's ascites hepatoma after 48 hr of starvation, and in ascites cells. The coefficient (K) showing the ratio of membrane-bound to free ribosomes was calculated. In the liver cells of healthy animals, the content of membrane-bound ribosomes was three times as high as that of free ribosomes ( $K=3$ ), but in ascites hepatoma cells, opposite ratios were observed. The content of free ribosomes was appreciably increased in the livers of animals with Zajdela's ascites hepatoma, and in the terminal stage (6 days) K approached 0.5. This value of the coefficient was characteristic of ascites cells. Analogous but less-pronounced changes were seen in the livers of control animals after 48 hr of starvation ( $K=0.89-1.0$ ). When rats with ascites tumors were force-fed, the relative content of free liver ribosomes remained high ( $K=0.78-1.3$ ), indicating that the tumor has a specific systemic effect on the liver, with or without the additive starvation. (18 refs.)
- 77-2369 Studies on the Mouse Liver h-Protein: Separation from Various Glutathione S-Transferases (Meeting Abstract).** (Eng.) Sarraf, A. M. (McArdle Lab. for Cancer Res., Univ. Wisconsin, Madison, WI 53706) McCarthy, K. L.; Heidelberger, C. *Fed Proc* 36(3): 865; 1977. (no refs.)
- 77-2370 Selective Phosphorylation of a Nuclear Envelope Polypeptide by an Endogenous Protein Kinase (Meeting Abstract).** (Eng.) Lam, K. S. (McArdle Lab. for Cancer Res., Univ. Wisconsin, Madison, WI 53706) Kasper, C. B. *Fed Proc* 36(3): 641; 1977. (no refs.)
- 77-2371 Phosphorylation of Nuclear and Plasma Membrane Proteins in Cultured Fibroblasts (Meeting Abstract).** (Eng.) Miller, M. R. (Sidney Farber Cancer Inst., Boston, MA 02115) Kletzien, R. F. *Fed Proc* 36(3): 640; 1977. (no refs.)
- 77-2372 Density-dependent Phosphorylation of a Specific Protein in Cultured Cells.** (Eng.) Wehner, M. (Dight Inst. Human Genetics, Univ. Minnesota, Minneapolis, MN 55455) Sheppard, J. R.; Malkinson, A. M. *Nature* 266(5605): 842-844; 1977.
- The phosphorylation of endogenous proteins in cultured mammalian cells at different cellular densities was studied to determine if cell-cell interactions affect protein phosphorylation. It was found that the phosphorylability of a specific protein present in several diverse cell lines exhibited density-dependence. The phosphorylability of this protein increased when the density of the cells in the plate was high, and was not dependent on experimental manipulations used to achieve high cellular density. The phosphorylation reaction was characterized. (21 refs.)
- 77-2373 Phosphoprotein Phosphatases in Rat Liver and Hepatoma (Meeting Abstract).** (Eng.) Farron-Furstenthal, F. (The Salk Inst., Box 1809, San Diego, CA) Lightholder, J. R. *Fed Proc* 36(3): 793; 1977. (no refs.)
- 77-2374 Evidence That Dephosphorylation Inactivates the Glucocorticoid Receptor of Mouse Fibroblasts (Meeting Abstract).** (Eng.) Nielsen, C. J. (Univ. Michigan Medical School, Ann Arbor, MI 48109) Sando, J. J.; Pratt, W. B. *Pharmacologist* 18(2): 326; 1976. (no refs.)
- 77-2375 Comparison of Glucocorticoid Receptors in Cytosols of Normal Liver, Liver of Tumor-bearing Organism, and Zajdela Hepatoma.** (Eng.) Dmitrieva, L. V. (Lab. Endocrinology, Biology Faculty, M. V. Lomonosov State Univ., Moscow, USSR) Volchek, A. G.; Rozen, V. B.; Adler, V. V.; Shapot, V. S. *Biochem* 41(10): 1502-1508; 1976.
- Specific protein receptors for two glucocorticoids, dexamethasone (D) and hydrocortisone (HC) were observed in the cytosols of cells of normal liver, hormone-resistant Zajdela ascites hepatoma, and the livers of tumor-bearing animals. Male rats of the Wistar line were utilized. A study of the competition of unlabeled hormones for the binding of  $^3\text{H}$ -D by the cell receptors of the livers of intact animals showed that the receptors had significant hormonal stereospecificity. The receptors of liver cell cytosol had an association constant of  $3.8 \times 10^8 \text{ M}^{-1}$  for D and  $0.57 \times 10^8 \text{ M}^{-1}$  for HC, as well as a limited number of steroid-binding sites. The steroid-receptor complexes of the cytosols of liver cells and ascites hepatoma sedimented in the 6S-7S region in a buffer of low ionic strength and in the 3S-4S region in a buffer of high ionic strength. The affinity of the receptors for HC and D changed as the Zajdela ascites hepatoma grew, and the number of steroid-binding sites declined three- to fourfold compared to

normal liver. This can partially explain the resistance of the hepatoma to glucocorticoids. (25 refs.)

**77-2376 Regulation of Glucocorticoid Receptor Levels in the AtT-20 Mouse Pituitary Tumor Cell by Corticosterone (Meeting Abstract).** (Eng.) Harrison, R. W. (Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232) *Fed Proc* 36(3): 2674; 1977. (no refs.)

**77-2377 Hormone Receptors Uncoupled from Adenylate Cyclase (AC) in a Variant S49 Lymphoma Cell Clone (Meeting Abstract).** (Eng.) Ross, E. M. (Dept. Pharmacology, Univ. Virginia, Charlottesville, VA 22903) Kaga, T.; Gilman, A. G. *Fed Proc* 36(3): 319; 1977. (no refs.)

**77-2378 Description of a New Hamster Ventral Prostate Cell Line Containing Androgen Receptors.** (Eng.) Norris, J. S. (Dept. Cell Biology and Medicine, Baylor Coll. Medicine, Houston, TX 77030) Bowden, C.; Kohler, P. O. *In Vitro* 13(2): 108-114; 1977.

Utilizing chemical transformation of primary monolayers of hamster ventral prostate (HVP) tissue, the HVP-G3 and HVP-B1 cloned cell lines were established. Phase contrast microscopy revealed different cell types in the two clones ranging from epithelioid to fibroblastic. The cell doubling time during log-phase growth for the HVP-G3 cells in Dulbecco's modified Eagle medium (DME) with 10% serum was 12.8 hr. Luteinizing hormone (LH, 5 µg/ml) decreased this time to 12.5 hr. In the presence of 5% serum, the doubling time increased to 17.8 hr. In Ham's nutrient mixture F-12, cell doubling time in 10% serum was 13.1 hr, but it was decreased to 11.8 hr when 5 µg/ml LH were included. Plating efficiency in F-12 ranged from 48.9% to 93%, compared to 63.9% to 93% in DME. LH (20 µg/ml) stimulated the HVP cells to incorporate <sup>3</sup>H-thymidine at the same rate as 10% serum. Neither cell line showed a growth response to androgens, but both contained high-affinity, saturable androgen receptors that migrated into the nucleus when they were exposed to physiological doses of androgen. When injected sc into male or female Syrian hamsters, each cell line produced a tumor in 6-8 wk. Both cell lines have a diploid stemline. (12 refs.)

**77-2379 Role of a Small Molecule Inhibiting Activation of Estrogen Receptor in Estrogen Action on Mouse Leydig Cell Tumors (Meeting Abstract).** (Eng.) Sato, B. (Dept. Biochemistry, Univ. Utah, Salt Lake City, UT 84132) Huseby, R. A.; Samuels, L. T. *Fed Proc* 36(3): 389; 1977. (no refs.)

**77-2380 Prolactin Binding in Rat Testis: Specific Receptors in Interstitial Cells.** (Eng.) Charreau, E. H. (Inst. Pathology, Rikshospitalet, Oslo, Norway) Attramadal, A.; Torjesen, P. A.; Purvis, K.; Calandra, R.; Hansson, V. *Molec Cell Endocrinol* 6(4-5): 303-307; 1977.

The localization of specific prolactin receptors in particular testicular cell types was attempted. Decapsulated, homogenized testes from adult Sprague-Dawley rats were incubated with iodinated human prolactin (PR), luteinizing hormone (LH), or growth hormone (GH). A gradual saturation of PR binding sites in testicular membranes reached a plateau at about 0.3 picomole (pmol) of PR after increasing amounts of labeled PR were added. Approx 1.5 nanograms of cold PR were required to elicit a 50% inhibition of added label. Regression analysis found about 0.020 pmol of PR binding sites per 100 mg of testes. The binding of LH was about 7 pmol compared to 0.33 pmol/testis for PR, with the binding of both hormones restricted to the interstitial compartment; tubular binding was negligible or absent. Only 55% of the PR receptors were recovered in the interstitial cell particles, compared to 75%-80% for LH, which suggests that PR has a higher receptor lability. PR and human GH were equally effective in displacing labeled PR from interstitial membrane receptors; ovine follicle-stimulating hormone and ovine LH were without effect. The data suggest that the PR effect on OH-stimulated testosterone secretion results from a direct PR stimulus of Leydig cells. (9 refs.)

**77-2381 Osmotic Control of the Release of Prolactin and Thyrotropin in Euthyroid Subjects and Patients with Pituitary Tumors.** (Eng.) Sowers, J. R. (Endocrine Section, Medical Res. Services, Veterans Admin. Wadsworth Hosp. Center, Los Angeles, CA) Hershman, J. M.; Skowsky, W. R.; Carlson, H. E.; Park, J. *Metabolism* 26(2): 187-192; 1977.

The influence of acute changes in serum osmolality on basal serum thyrotropin (TSH) and prolactin (PRL) levels and on the responses of TSH and PRL to the TSH-releasing hormone (TRH) analog, N3im-methyl-TRH, was assessed in 10 euthyroid subjects and in 3 patients with PRL-secreting pituitary tumors. The results suggest that changes in osmolality in euthyroid patients may have a direct effect on the PRL and TSH response to a releasing factor. (17 refs.)

**77-2382 Comparison of the In Vitro Conversion of Estradiol-17β to Estrone of Normal and Neoplastic Human Breast Tissue.** (Eng.) Pollow, K. (Inst. für Molekularbiologie und Biochemie, Frauenklinik Charlottenburg der Freien Univ. Berlin, 1000 Berlin 33, W. Germany) Boquoi, E.; Baumann, J.; Schmidt-Gollwitzer, M.; Pollow, B. *Mol Cell Endocrinol* 6(4-5): 333-348; 1977.

The activity of 17β-hydroxysteroid dehydrogenase (17β-



HSD), which catalyses the conversion of estradiol-17 $\beta$  to estrone in various subcellular fractions of nonmalignant and neoplastic breast tissues was measured. The activity was also correlated to the stage of the menstrual cycle in 35 tissue specimens of premenopausal women. Forty-eight breast cancer tissue specimens and 48 nonmalignant specimens were studied. The results showed a greater conversion of estradiol-17 $\beta$  into estrone with the nonmalignant tissues. Specific enzyme activity depended on the phase of the menstrual cycle, with the highest 17 $\beta$ -ASD activity observed with subcellular fractions obtained during the early secretory phase rather than during the proliferation phase. (36 refs.)

- 77-2383 Functional Activity of a Gonadotrophin-Producing Human Lung Carcinoma After Transplantation in Nude Mice.** (Eng.) Sindelar, W. F. (Dept. Surgery, Univ. Maryland Sch. Medicine, Baltimore, MD) Liebllich, J. M.; Rosen, S. W.; Tralka, T. S.; Rabson, A. S. *Surg Forum* 27: 84-86; 1976.

The retention of both structural and functional characteristics of a gonadotrophin-secreting human lung carcinoma following transplantation into nude mice was investigated. ChaGo cells were harvested mechanically from culture vessels and injected sc into the infrascapular regions of nude mice. A dose-response experiment established  $1.0 \times 10^7$  cells as the optimum inoculum. A total of 34 mice was inoculated, with 24 producing solid tumors at the injection sites. Tumors from four animals were transplanted serially in solid form for more than four generations. Tissue sections prepared from heterotransplanted first-generation tumors in nude mice were compared with sections taken from the lymph node biopsy originally utilized to initiate the ChaGo cultures. Both the transplanted and original tumors showed identical morphologic findings: a highly cellular malignant stroma and numerous bizarre giant cells. Sections were prepared for transmission electron microscopy from ChaGo cultures and from transplanted tumors. Similar ultrastructural features were present in both cultured cells and solid neoplasms. Specific radioimmunoassays were performed for human chorionic gonadotrophin (HCG)- $\alpha$  and whole HCG in the serum of tumor-bearing nude mice. Samples were obtained from 15 animals with first-generation neoplasms. The assays demonstrated wide variation but consistently elevated gonadotrophins. HCG- $\alpha$  ranged from 0.7-180 nanograms (ng)/ml, with higher levels occurring in animals bearing larger tumors. Non-tumor-bearing control mice had no detectable serum gonadotrophin. At 7 days after cell inoculation, pooled sera from five tumor-bearing mice showed an HCG- $\alpha$  level of 0.7 ng/ml; at 14 days, five animals had 1.2-11.0 ng/ml; at 21 days, one animal had 15.8 ng/ml; at 28 days, two mice had 8.4 and 180 ng/ml; and at 42 days, two tumor-bearing animals had 1.4 and 40 ng/ml. Host serum continued to contain elevated HCG- $\alpha$  after serial transplantation of solid tumors. In tumor transplant line 1, HCG- $\alpha$  levels were 2.2 ng/ml in generation 1, 2.2 ng/ml in generation 2, and 1.8 ng/ml in generation 4. Uterine wts were increased in tumor-bearing

mice, averaging 181 ng in 24 experimental animals compared to 140 ng in 10 non-tumor-bearing controls. The nude mouse is a satisfactory model for studying behavior of ectopically functioning human neoplasms. (2 refs.)

- 77-2384 Prostaglandin Synthesis and Prostaglandin E-9-Ketoreductase in Normal and Neoplastic Rat Mammary Gland (Meeting Abstract).** (Eng.) Carpenter, M. P. (Biomembrane Res. Lab., Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104) Robinson, R. D.; Thuy, L. P. *Fed Proc* 36(3): 767; 1977. (no refs.)

- 77-2385 Hexosamine-Containing Macromolecules in Human Colon Carcinomas.** (Eng.) Terho, T. (Dept. Physiology, Univ. Turku, Turku, Finland) Laitio, M. *Scand J Gastroenterol* 12(1): 7-15; 1977.

The normal, transitional, and malignant areas of five colons resected for carcinoma were examined morphologically, histochemically, and biochemically to isolate and characterize the hexosamine-containing macromolecules in each area. Sulfated mucin was predominant in normal mucosa, but the transitional areas showed a predominance of nonsulfated acid mucin. In malignant areas the secretion was composed almost entirely of acid nonsulfated mucosubstances or mixtures of acid nonsulfated and neutral mucins. The concentration of total hexosamine-containing macromolecules was 1.5 times higher in the transitional areas and 2 times higher in the carcinomas than in the normal mucosa. These macromolecules were divided into three groups: (1) acid glycosaminoglycans; (2) high molecular weight (MW) glycopeptides, and (3) low MW glycopeptides. The concentration of the saline-insoluble fraction of the low MW glycopeptides was 2 times higher in transitional areas and 4 times higher in carcinoma areas than in normal mucosa. It had been previously suggested that the acid glycosaminoglycans might be able to form a coat around malignant cells, impeding contact of immunologically active cells with the neoplasm and preventing immunological reactions against it. Thus, acid glycosaminoglycans might serve as a ready-growth floor for tumor spread. (33 refs.)

- 77-2386 Glycopeptides and Glycosaminoglycans of Human Mammary Cells (Meeting Abstract).** (Eng.) Chandrasekaran, E. V. (M. S. Hershey Medical Center, Pennsylvania State Univ., Hershey, PA 17033) Banks, J.; Davidson, E. A. *Fed Proc* 36(3): 644; 1977. (no refs.)

- 77-2387 Partial Structure of Surface Membrane Glycopeptides (Meeting Abstract).** (Eng.) Santer, U. V. (Univ. Pennsylvania Medical Sch., Children's Hosp. Philadelphia, Philadelphia, PA 19104) Glick, M. C. *Fed Proc* 36(3): 746; 1977. (no refs.)

77-2388 **Glycoconjugate Biosynthesis in Closely Related Mouse Fibroblasts in Tissue Culture (Meeting Abstract).** (Eng.) Winterbourne, D. J. (NIH, Bethesda, MD 20014) *Fed Proc* 36(3): 643; 1977. (no refs.)

77-2389 **Blood Group Glycolipid Biosynthesis in Tumor and Differentiated Cultured Cells (Meeting Abstract).** (Eng.) Presper, K. A. (Dept. Chemistry, Univ. Notre Dame, Notre Dame, IN 46556) Basu, M.; Basu, S. *Fed Proc* 36(3): 731; 1977. (no refs.)

77-2390 **Lipid Metabolism in HeLa Cells Inhibited by Pentadecan-2-One (Meeting Abstract).** (Eng.) Naccarato, W. F. (Univ. Pittsburgh Sch. Dental Medicine, Pittsburgh, PA 15261) Gilbertson, J. R.; Fletcher, R. D. *Fed Proc* 36(3): 773; 1977. (no refs.)

77-2391 **Exchange of Phospholipids Between Mitochondria and Microsomes of Rat Hepatoma 27.** (Eng.) Dyatlovitskaya, E. V. (M. M. Shemyakin Inst. Bioorganic Chemistry, Acad. Sciences USSR, Moscow, USSR) Timofeeva, N. G.; Gor'kova, N. P.; Bergel'son, L. D. *Biochemistry (Translation)* 41(7): 1009-1013; 1976.

The exchange of phospholipids between isolated mitochondria and microsomes of rat hepatoma 27 and normal rat liver during incubation with the postmitochondrial supernatant was studied. Hepatoma 27 was obtained on the 21st day after tumor transplantation. Labeled subcellular fractions were obtained by injecting  $^{32}\text{P}$  ip on the 18th day after transplantation. Mitochondria and microsomes of the liver and hepatoma 27 were isolated by differential centrifugation. The labeled (unlabeled) mitochondria were incubated with unlabeled (labeled) microsomes of the hepatoma for 5, 15, 30, and 60 min; the liver mitochondria and microsomes were incubated for 30 and 60 min. The phospholipids were extracted from the subcellular fractions and identified by thin layer chromatography. Intermembrane exchange of the phospholipids was substantially activated when the mitochondria and microsomes of the hepatoma were incubated with the supernatant. Phosphatidylcholine was the most intensively exchanged from mitochondria to microsomes, followed by phosphatidylserine and phosphatidylinositol, and then phosphatidylethanolamine and sphingomyelin. Cardiolipin was the most inert phospholipid. The transfer of phospholipids from the microsomes to the mitochondria proceeded less intensively, with the exception of cardiolipin. The exchange of phospholipids between subcellular fractions of the liver occurred more slowly than in the hepatoma. The data indicate that the previously detected lipid dedifferentiation of the membranes in tumor cells may be associated with disruptions

of the intermembrane exchange of phospholipids. It must still be determined whether these disturbances are due to changes in the membrane structure or to changes in the activity and specificity of the lipid-exchanging proteins of the tumor cells. (16 refs.)

77-2392 **Octadecenoate Isomers at the 1- and 2-Positions of Hepatoma and Liver Phospholipids (Meeting Abstract).** (Eng.) Wood, R. (Dept. Biochemistry, Texas A&M Univ., College Station, TX 77843) Chumbler, F. *Fed Proc* 36(3): 852; 1977. (no refs.)

77-2393 **Phospholipid Composition of Rat Liver and Hepatoma 27 Nuclear Membranes and Nuclei.** (Eng.) Lemenovskaya, A. F. (M. M. Shemyakin Inst. Bioorganic Chemistry, Acad. Sciences USSR, Moscow, USSR) Keon, Y. M.; Perevoshchikova, K. A.; Zbarskii, I. B.; Dyatlovitskaya, E. V.; Bergel'son, L. D. *Exp Biol Med* 81(5): 818-821; 1976.

The phospholipid composition of rat liver and hepatoma 27 nuclei and nuclear membranes is evaluated. Rat liver nuclear membranes and nuclei differed only in the content of choline-containing lipids. The amount of lecithin was decreased and the content of sphingomyelin was increased in the membranes as compared with isolated nuclei. Hepatoma nuclei and nuclear membranes differed from the corresponding subcellular fractions of the liver in the presence of a large amount of cardiolipin, a decreased content of phosphatidylcholine, and an increased content of sphingomyelin. In level of sphingomyelin, hepatoma nuclear membranes were similar to plasma membrane, the subcellular fraction being richest in sphingomyelin. A significant property of hepatoma nuclear membranes was that they contained twice as much acid phospholipids as liver nuclear membranes. Preparations of the nuclei and nuclear membranes of tumor cells differed only slightly in activity of the marker enzymes from the corresponding preparations obtained from the liver. The'-nucleotidase activity in hepatoma nuclear membranes was the same as in liver nuclear membranes, while the inosine diphosphatase activity was even lower. The succinate dehydrogenase activity in liver and hepatoma nuclear membranes comprised 19% and 49% of the activity in the mitochondrial fraction. Even if the admixtures of mitochondria comprised 50% in a preparation of hepatoma nuclear membranes, the amount of cardiolipin could not exceed 3%. Hepatoma nuclear membranes contained their own intrinsic cardiolipin. A high content of sphingomyelin was characteristic of native hepatoma nuclear membranes. The phenomenon of lipid dedifferentiation of the membranes, found earlier for the mitochondria, microsomes, and plasmatic membranes of tumor cells, also extends to the nuclear membranes of hepatoma cells. (14 refs.)



- 77-2394 **The Influence of Lipid Composition on Some Physical and Functional Properties of Cellular Membranes in Rat Liver and Hepatomas.** (Eng.) Feo, F. (Istituto de Patologia Generale, Corso Raffaello 30, 10125 Torino, Italy) Canuto, R. A.; Garcea, R.; Gabriel, L. *Panminerva Med* 18(11/12): 454-471; 1976.

The effect of lipid composition on physical and functional properties of cellular membranes in rat liver and hepatomas is investigated. Respiratory rates, acceptor control and ADP:oxygen ratios of the same order occurred in mitochondria from adult liver, cholesterol-enriched liver, and hepatoma AH-130. The respiratory rates of mitochondria from fetal liver and hepatomas 3924A and 5123 were lower than those of adult rat liver mitochondria. An increase in free cholesterol content of four-to-six-fold was observed in mitochondria from cholesterol-enriched liver and hepatomas. In mitochondria from hepatomas, changes in fatty acid composition similar to those noted in fetal liver occurred: myristate, palmitoleate and oleate increased, while arachidonate decreased. Changes in mitochondrial water content during  $K^+$  uptake were determined in adult rat liver and hepatoma AH-130 mitochondria. Total water increased 20% in both rat liver and hepatoma AH-130 mitochondria. The phospholipid to cholesterol ratios in microsomes and mitochondria from hepatomas 5123, AH-130, and 3924A were lower than in the particles isolated from fetal or adult rat livers. The in vivo enrichment of rat liver with cholesterol resulted in 1.5- and 3.7-fold increases of cholesterol content in microsomes and mitochondria, respectively. There occurred a decrease in the extent of phosphate-induced and hypoosmotic swelling in mitochondria from hepatomas and cholesterol-enriched liver. The cholesterol content did not influence  $K^+$  uptake by hepatoma and cholesterol-enriched mitochondria. Changes in lipid fluidity should be taken into account in pathological processes in which biological membranes are involved. (82 refs.)

- 77-2395 **Synthesis of Surface-Active Phospholipids by a Cell Culture of a Combined Cell Carcinoma of the Lung from a Beagle Dog (Meeting Abstract).** (Eng.) Pfleger, R. C. (Inhalation Toxicology Res. Inst., Albuquerque, NM 37115) Hahn, F. F.; Lay, J. C. *Fed Proc* 36(3): 790; 1977. (no refs.)

- 77-2396 **Effects of Altering Diet Fat on Breast Fluid Lipids in Women (Meeting Abstract).** (Eng.) Petrakis, N. L. (Univ. California, San Francisco, CA 94143) Mason, M. L.; Doherty, M.; Dupuy, M. E.; Sadee, C.; Wilson, C. S. *Fed Proc* 36(3): 1163; 1977. (no refs.)

- 77-2397 **Membrane Saccharides of Rat Liver and Malignant-Cell Nuclei.** (Eng.) Stoddart, R. W. (Strangeways Res. Lab., Worts' Causeway, Cambridge CB1 4RN, England) *Biochem Soc Trans* 5(1): 121-122; 1977.

To study differences between normal and neoplastic nuclear membranes, nuclei were isolated from normal Wistar rat liver and two transplanted rat tumors, a 4-dimethylaminoazobenzene-induced hepatoma and a 3-methylcholanthrene-induced fibrosarcoma. The findings indicate that it is possible to distinguish differences in the carbohydrate profile of the nuclear membranes from normal and malignant rat tissues; sialyl and D-galactose-like residues, reactive with aprotinin and *Ricinus communis* agglutinin, were decreased in the sarcoma and hepatoma nuclear preparations as compared with those of normal liver preparations. Further studies are necessary to obtain a more precise localization of specific sugar residues in the nuclear membrane, since in the initial isolation of nuclei, some elements of the outer nuclear membrane may be lost, thus exposing regions of the inner nuclear membrane. (6 refs.)

- 77-2398 **Role of Diet on Composition of Rat Bile (Meeting Abstract).** (Eng.) Davis, J. W. (St. Louis Univ. Sch. Medicine, St. Louis, MO 63104) Elliott, W. H.; Foelsch, J. M.; Ruminski, P. *Fed Proc* 36(3): 1143; 1977. (no refs.)

- 77-2399 **Characterization of a Collagenous Protein Secreted by Murine Teratocarcinoma Cells in Culture (Meeting Abstract).** (Eng.) Ko, C. (Dept. Pathology, Emory Univ., Atlanta, GA 30322) Johnson, L. D. *Fed Proc* 36(3): 1069; 1977. (no refs.)

- 77-2400 **Collagen Synthesis as a Marker for Cell Type in Mouse 3T3 Lines.** (Eng.) Goldberg, B. (Dept. Pathology, New York Univ. Medical Center, New York, NY 10016) *Cell* 11(1): 169-172; 1977.

Collagen synthesis was used to determine the fibroblastic or endothelial origin of Swiss/3T3 and Balb/3T3 (clone A31) cell lines. The amounts and types of collagen produced were compared. Both Swiss and Balb/3T3 cultures synthesized only collagen types I and III, thus indicating a fibroblastic origin and function. (26 refs.)

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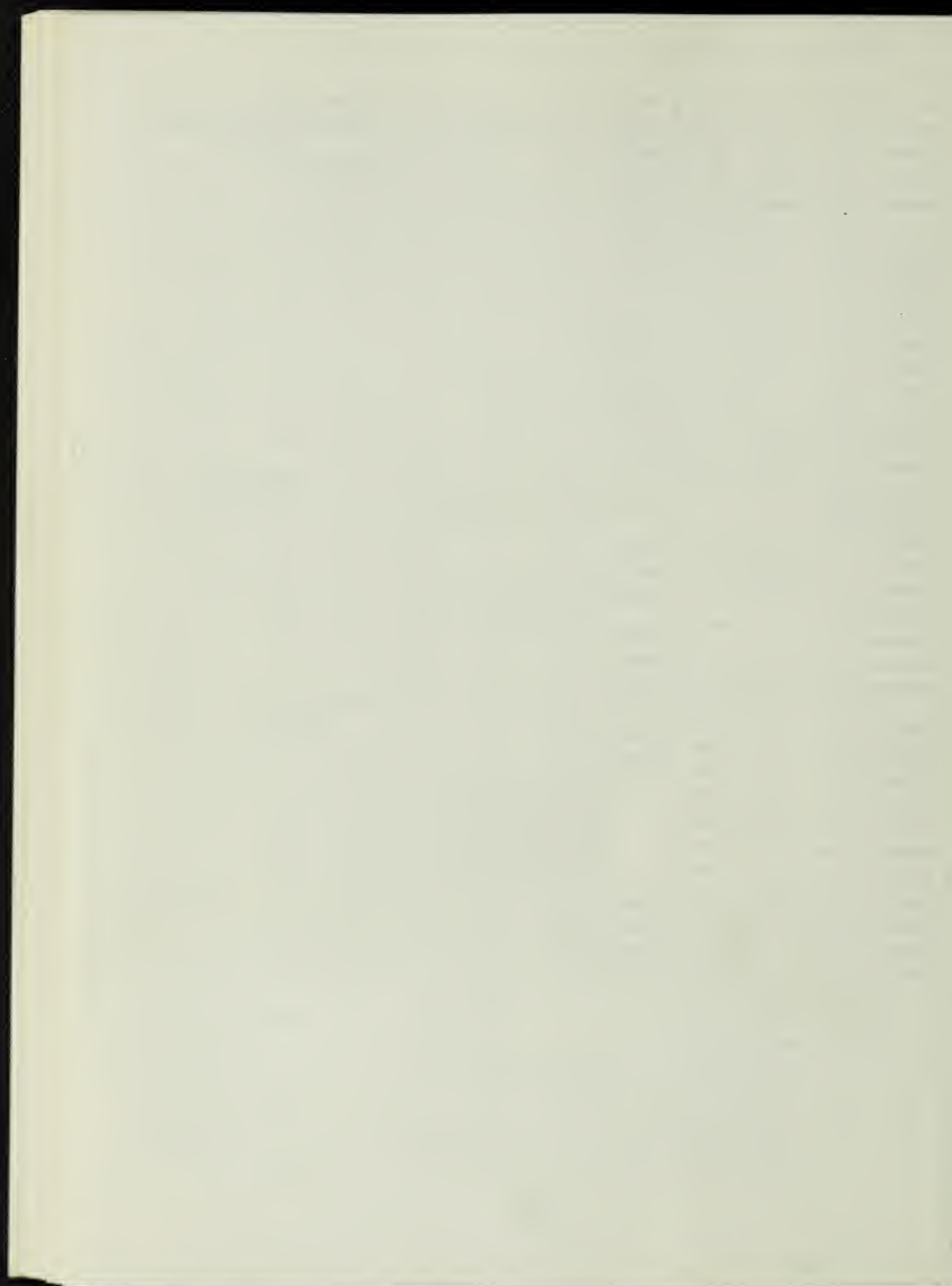
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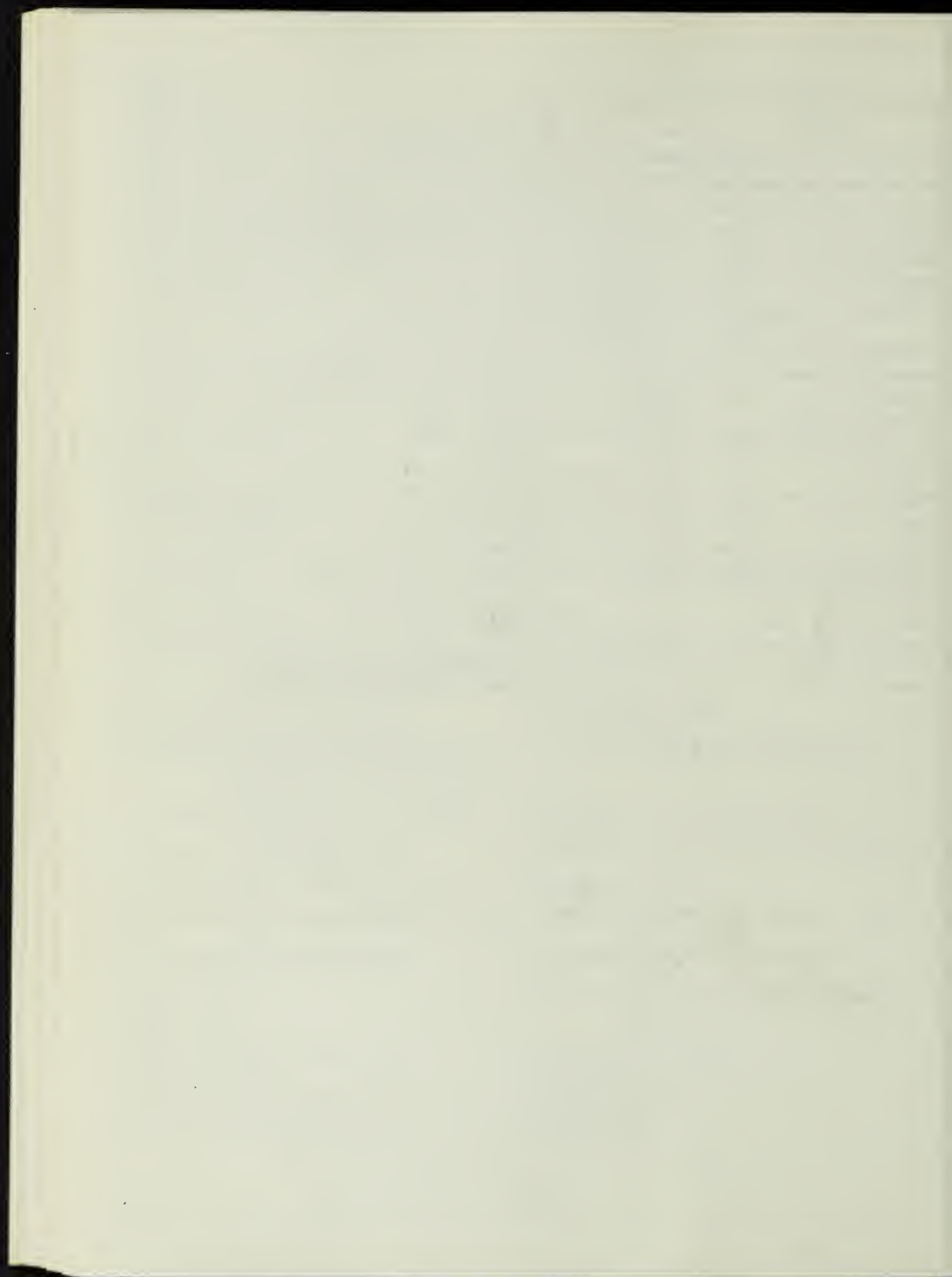
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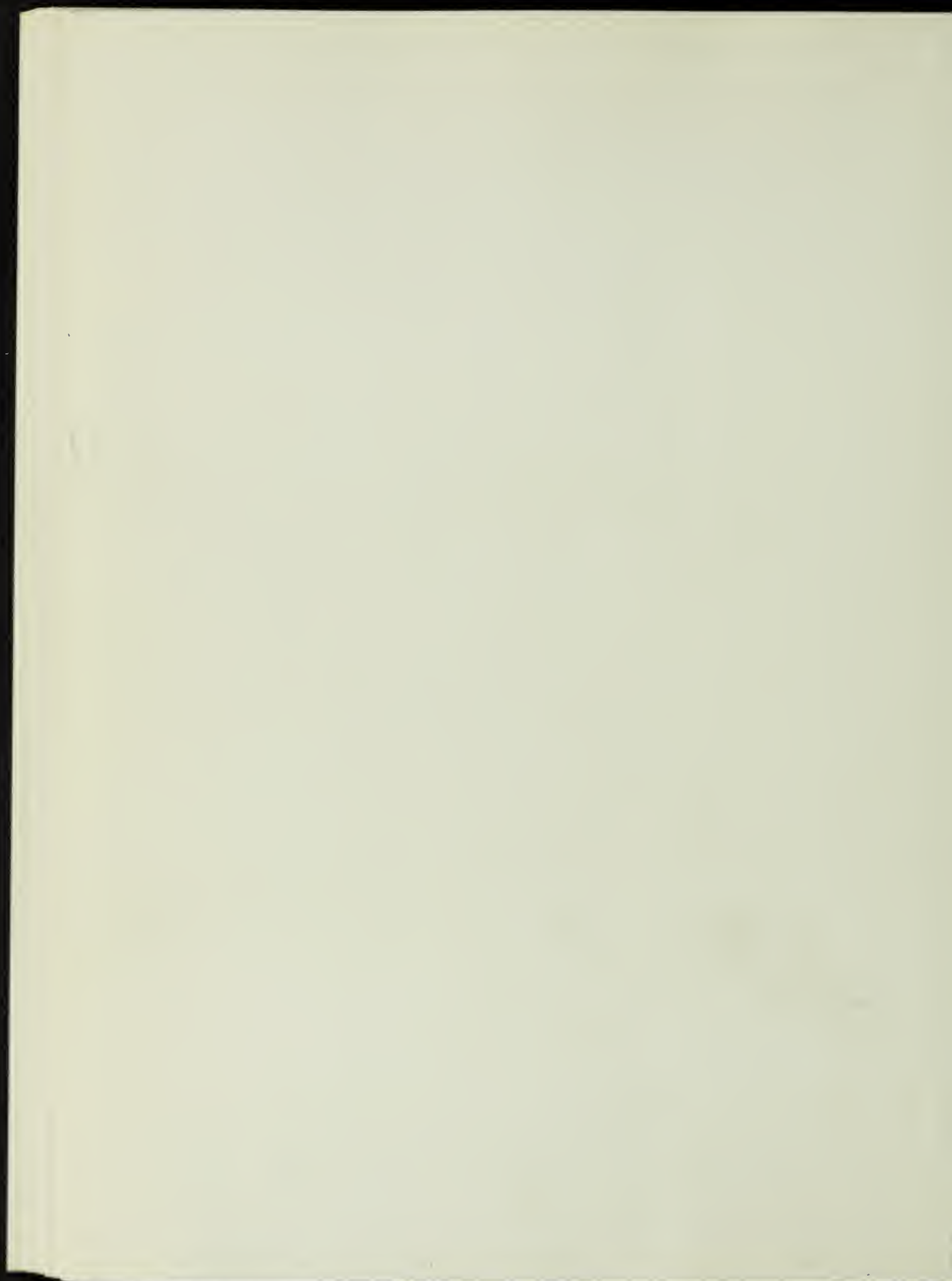
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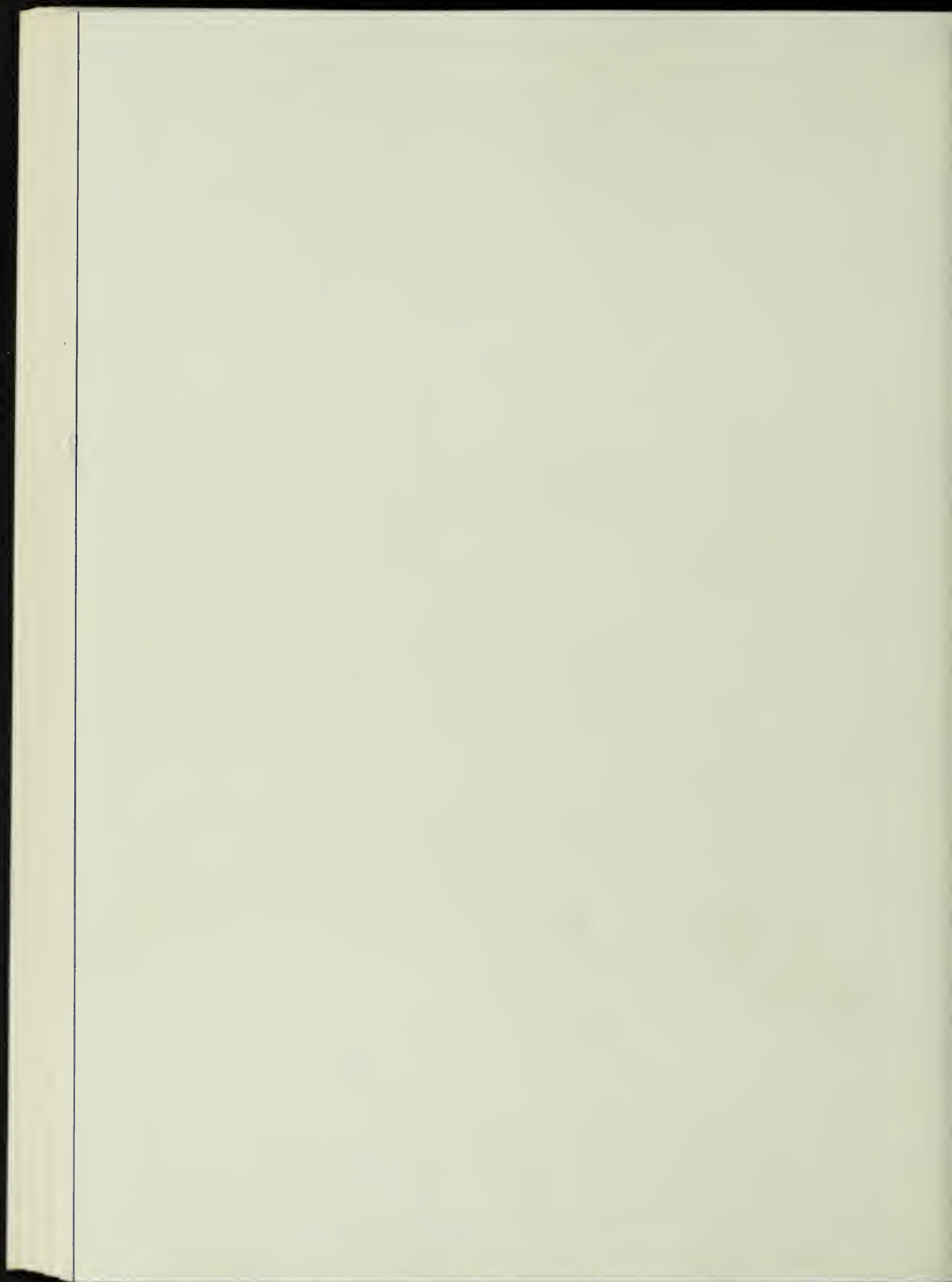
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## ABBREVIATIONS

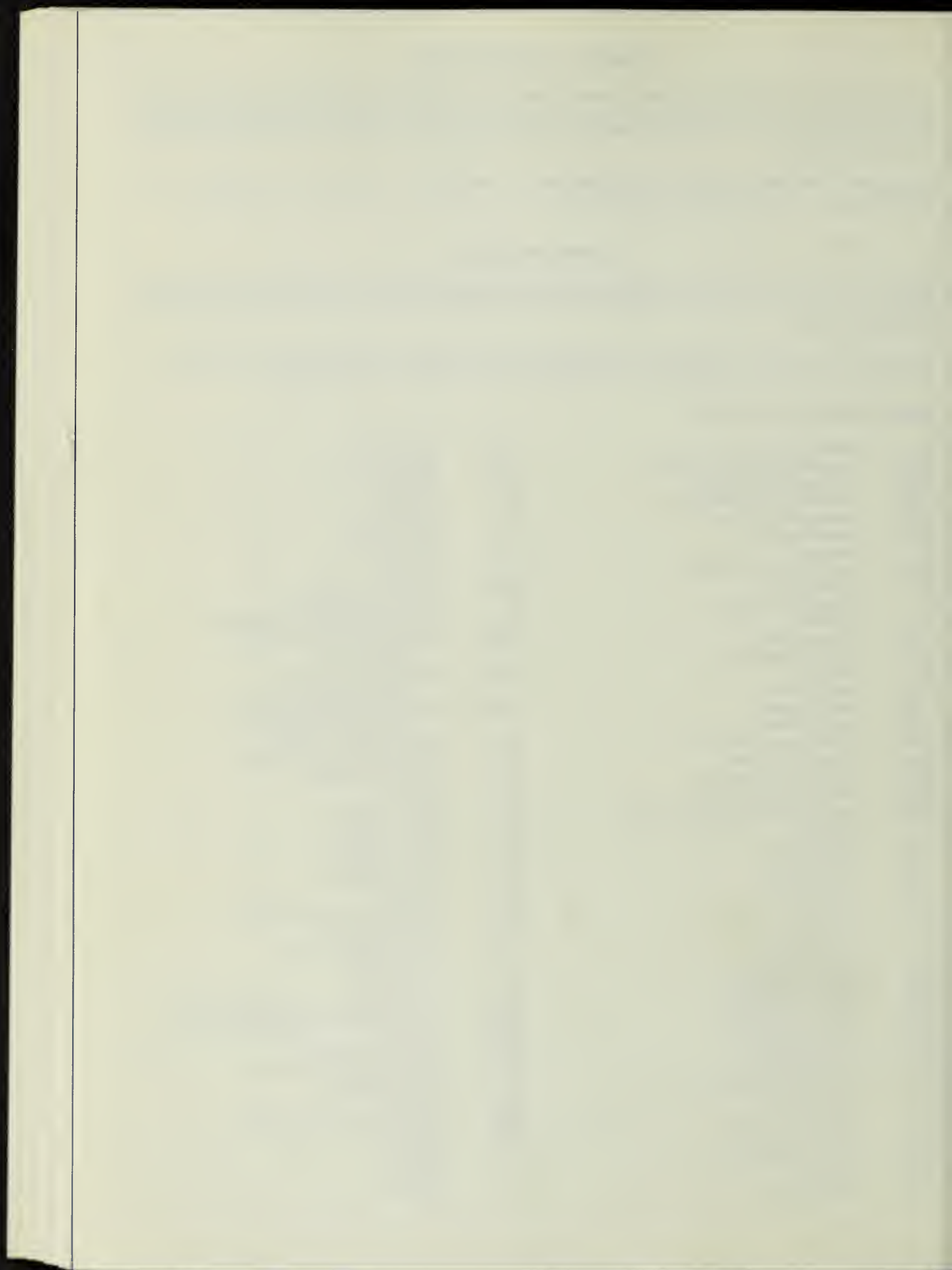
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**ABBREVIATIONS** used in abstracts:

A	angstrom(s)	mOsm	milliosmolar
ACTH	adrenocorticotrophic hormone	max	maximum
ADP	adenosine diphosphate	mEq	milliequivalent(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
approx	approximately	μl	microliter(s)
av	average	mm	millimeter(s)
BCG	bacillus Calmette-Guerin	mo	month(s)
bid	twice daily	mol wt	molecular weight
C	degree(s) centigrade	N	normal concentration
cal	calorie(s)	NAD	nicotinamide adenine dinucleotide
kcal	kilocalorie(s)	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
Ci	curie(s)	NADPH	reduced nicotinamide adenine dinucleotidephosphate
mCi	millicurie(s)	NCI	National Cancer Institute
μCi	microcurie(s)	NIH	National Institutes of Health
cm	centimeter(s)	PAS	periodic acid-Schiff
CNS	central nervous system	po	orally
cpm	counts per minute	ppb	parts per billion
DNA	deoxyribonucleic acid	ppm	parts per million
ED <sub>50</sub>	median effective dose	qid	four times daily
EDTA	ethylenediamine tetraacetic acid	qod	every other day
g	gram(s)	QO <sub>2</sub>	oxygen quotient
kg	kilogram(s)	R	roentgen
mg	milligram(s)	RBC	red blood cells (erythrocytes)
μg	microgram(s)	RNA	ribonucleic acid
Hb	hemoglobin	rpm	revolutions per minute
hr	hour(s)	sc	subcutaneous
ia	intra-arterial	sec	second(s)
id	intra-dermal	SGOT	serum glutamic-oxaloacetic transaminase
IgA	Immunoglobulin A	SGPT	serum glutamic-pyruvic transaminase
IgB	Immunoglobulin B	soln	solution
IgG	Immunoglobulin G	TCD	tissue culture dose
IgM	Immunoglobulin M	TCD <sub>50</sub>	median tissue culture dose
ILS	increased life span	tid	three times daily
im	intramuscular	UV	ultraviolet
ip	intraperitoneal	WBC	white blood cells (leukocytes)
IU	International Unit(s)	wk	week(s)
iv	intravenous	wt	weight
Km	Michaelis constant	X	times
LD	lethal dose	yr	year(s)
LD <sub>50</sub>	median lethal dose		
M	molar		
μM	micromolar		





## REVIEW

**77-2401 Androgen Receptors in Male Sex Tissues of Rats and Humans.** (Eng.) Attramadal, A.; Weddington, S. C.; Naess, O.; Djoseland, O.; Hansson, V. In: *Prostatic Disease. Proceedings of the American-European Symposium held in Vienna, November 3-5, 1975.* Marberger, H.; Haschek, H.; Schirmer, H. K.; Colston, J. A.; Witkin, E., eds. (New York: Alan R. Liss, Inc.): pp. 189-203; 1976.

The physicochemical properties of the androgen receptors in various target tissues are described. Sucrose gradient centrifugation has shown that the androgen receptors sediment as 7S-8S complexes at low ionic strength and as 4S complexes at high ionic strength. The receptors are heat-sensitive and are completely destroyed at temperatures  $> 40^{\circ}\text{C}$ . All the androgen receptors studied were able to bind biologically active steroids and were sensitive to sulphydryl reagents. The equilibrium constant of association was found to be  $10^{10} \text{ M}^{-1}$ . The dissociation of bound androgens at  $0^{\circ}\text{C}$  was very slow. Sephadex gel chromatography revealed that the receptors have a Stokes radius  $> 80 \text{ \AA}$ . Receptors in all tissues studied showed the same electrophoretic mobility in agarose acrylamide gels and the same isoelectric point ( $\text{pI} = 5.8$ ). Androgen receptors in the prostate, seminal vesicles, and epididymis were able to bind  $5\alpha$ -dihydrotestosterone (DHT) better than testosterone. Receptors in the testis, submaxillary glands, kidney, levator ani muscle, pituitary, ovary, and uterus bound testosterone as well as or better than DHT. Human androgen receptors were destroyed by protease but not by RNase or DNase. No physicochemical differences between receptors in prostatic hyperplasia and cancerous tissue were demonstrated. Androgen receptors in all organs and species are similar if not identical. Differences in steroid binding are due to organ-specific differences in target tissue metabolism rather than to differences in steroid specificity of the receptors. (46 refs.)

**77-2402 Hormone Replacement Therapy and Endometrial Carcinoma (Letter to Editor).** (Eng.) Doll, R. (Dept. Regius Professor Medicine, Univ. Oxford, Oxford OX2 6HE, England) Kinlen, L. J.; Skegg, D. C.; Smith, P. G.; Vessey, M. P. *Lancet* 1(8014): 745; 1977.

A reply is made to the dismissal of epidemiological evidence as "fragile" that estrogen treatment increases the risk of endometrial cancer. Notable increases in cancer of the corpus uteri have been recorded in several US cancer registries from 1969 to 1973. In addition, the most striking increases were

seen in the age group 55-64 yr, in which any effect of the comparatively recent growth in the use of hormone replacement therapy would be expected. Similar changes have not been observed in the UK, but estrogens have been prescribed there on a much smaller scale than in the US. The relative risks for endometrial cancer associated with the use of hormone replacement therapy were estimated from three recent retrospective studies to be between 4 and 8 to 1. Epidemiological evidence does not prove a causal relationship, but it is strongly suggestive of one. (7 refs.)

**77-2403 The Role of Prolactin in Carcinogenesis.** (Eng.) Kim, U.; Furth, J. In: *Vitamins and Hormones. Advances in Research and Applications, Vol. 34.* Munson, P. L.; Glover, J.; Diczfalussy, E.; Olson, R. E., eds. (New York: Academic Press): pp. 107-136; 1976.

The role of prolactin in carcinogenesis is described. Epidemiological studies of mammary cancer in different races suggest the existence of genetic differences in man, but they are often obscured by demographic differences. Some have postulated differences in estrogen or androgen metabolism, while others have presumed high risk factors because plasma prolactin is elevated in near relatives of breast cancer patients. The susceptibility of mice to mammary tumor virus is highest at birth and during the nursing period and decreases with age. High dietary fat increases mammary tumor incidence in rats given chemical carcinogens. This has been attributed to elevated serum prolactin levels. Under estrogenic stimulation, the mammatropes promptly undergo hyperplasia, with increased secretion of prolactin. This is a reversible process. Regression of hypersecretion of prolactin follows cessation of estrogenic stimulation. However, if the estrogenic stimulation is uninterrupted, some prolactin cells can undergo neoplastic transformation followed by formation of prolactin-secreting adenomas. The discovery of the hypothalamic hormones and agents acting by way of the hypothalamus has emphasized the role of prolactin in mammary carcinogenesis. The triggering mechanism of mammary tumorigenesis can lie in the CNS. Prolactin appears to be the key hormone, although estrogens also are frequently involved. Prolactin acts on the plasma membrane of the mammary gland, where its instructions for growth and function of the mammary epithelium are carried out by cyclic AMP. The fact that mammary tumors cannot be induced in hypophysectomized animals in the absence of prolactin calls for reconsideration of the view that the major direct mammary carcinogens are ovarian steroids. (233 refs.)



- 77-2404 **Hepatotoxicity and the Oral Contraceptives** (Letter to Editor). (Eng.) O'Connor, J. A. (477 North Harbor Drive, Indian Rocks Beach, FL 33535) *Ann Intern Med* 86(1): 118-119; 1977.

All oral contraceptives currently in use consist of 17 $\alpha$ -ethynyl substituted compounds, which do not occur naturally in human biochemistry and therefore are not readily metabolized. (1 ref.)

- 77-2405 **Estrogen Revisited.** (Eng.) Gusberg, S. B. (Dept. Obstetrics and Gynecology, Mount Sinai Sch. Medicine City Univ. New York, New York, NY) *J Reprod Med* 18(6): 325-326; 1977.

Evidence is given that endometrial stimulation by estrogen at a continuous rate without modification can induce endometrial cancer in susceptible women. The value of estrogen in postmenopausal women appears to be limited to the control of dyspareunia. (11 refs.)

- 77-2406 **Estrogens and Reproductive Cancer: An Invitational Symposium.** (Eng.) Zuspan, F. P. (Dept. Obstetrics and Gynecology, Ohio State Univ., Columbus, OH) *J Reprod Med* 18(6): 285-286; 1977.

Various approaches to the use of estrogen therapy and its causative relationship to endometrial cancer in postmenopausal women are briefly summarized. (1 ref.)

- 77-2407 **Estrogen and Endometrial Carcinoma.** (Eng.) Knab, D. R. (Dept. Obstetrics and Gynecology, Uniformed Services Univ. Health Sciences, Bethesda, MD 20014) *Obstet Gynecol Surv* 32(5): 267-281; 1977.

The literature implicating estrogen in the development of endometrial cancer is reviewed. Evidence is presented that adrenal androgen production of androstenedione, associated with a normal to increased peripheral conversion to estrone, may create a hormonal milieu that can lead to endometrial carcinoma in a susceptible patient. (187 refs.)

- 77-2408 **Another Risk of Phenacetin Containing Analgesic.** (Eng.) Anonymous (No affiliation given) *NZ Med J* 85(586): 338; 1977.

Literature is reviewed on tumors of the renal pelvis associated with excessive use of analgesics containing phenacetin. Due

to the possible carcinogenic properties of phenacetin metabolites, analgesics containing this drug have been banned in New Zealand. (10 refs.)

- 77-2409 **Saccharin--The Bitter Sweet.** (Eng.) Isselbacher, K. J. (Harvard Medical Sch., Boston, MA 02115) Cole, P. *N Engl J Med* 296(23): 1348-1350; 1977.

Arguments are presented against banning the use of saccharin. Recent studies have not shown the carcinogenic properties of saccharin in humans. It is recommended that legislation be changed to guard against inappropriate extrapolation to man of experimental evidence from other species, and to permit a balancing of the benefits of a food additive against its dangers. (14 refs.)

- 77-2410 **Are Alcoholic Beverages Carcinogenic?** (Eng.) Wydner, E. L. (American Health Foundation, Valhalla, NY) *JAMA* 237(13): 1377; 1977.

Although there is a higher incidence of hepatocellular carcinomas and an increased risk of cancer of the oral cavity, larynx and esophagus in heavy drinkers, there is no definite correlation between the alcohol and cancer. Part of the problem may be the nutritional deficiencies associated with alcoholism. (2 refs.)

- 77-2411 **Antioxidants vs. Aging.** (Eng.) Packer, L. (Dept. Physiology, Univ. California at Berkeley, Berkeley, CA 94720) Walton, J. *Chemtech* 7(5): 276-281; 1977.

Since many chemical carcinogens and/or their intermediates may be free radicals or may be activated by free radicals, the consumption of antioxidants may lead to a decrease in cancer. This has already been proven in animal studies. The role of free radicals in aging is also discussed. (25 refs.)

- 77-2412 **Transplacental Transfer of Foreign Compounds and Their Metabolism by the Fetus.** (Eng.) Pelkonen, O. In: *Progress in Drug Metabolism*. Bridges, J. W.; Chasseaud, L. F. (London: John Wiley & Sons) Vol. 2, pp. 119-161; 1977.

The ability of the human fetus to metabolize xenobiotic compounds is reviewed. The fetal ability to oxidize these compounds appears to parallel the active steroid-hydroxylation characteristic of the fetus. Consequences of the metabolic process include accumulation of water-soluble metabolites and toxic intermediates, and steroid interactions. (241 refs.)

- 77-2413 **The Metabolism of Xenobiotics in Cell Suspensions and Cell Cultures.** (Eng.) Fry, J. R.; Bridges, J. W. In: *Progress in Drug Metabolism*. Bridges, J. W.; Chasseaud, L. F. (London: John Wiley & Sons): Vol. 2, pp. 71-118; 1977.

The use of various culture and cell suspension techniques for the study of compounds foreign to animal cells, including polycyclic hydrocarbons, is discussed. Emphasis is placed not only on the xenobiotic metabolism in freshly isolated cells but also on the metabolism in cultured cells. (226 refs.)

- 77-2414 **Pronetalol Hydrochloride.** (Eng.) IARC Working Group In: *IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Some Miscellaneous Pharmaceutical Substances*. International Agency for Research on Cancer (Lyon, France): Vol. 13, pp. 227-231; 1977.

Chemical and physical data; production, use, occurrence, and analysis; and available metabolic, mutagenic, toxicological, and carcinogenic data are given for pronetalol hydrochloride (PH:  $\alpha$ -[(1-methylethyl)amino] methyl) -2- naphthalene-methanol, hydrochloride), the salt of pronetalol, a potent  $\beta$ -adrenergic blocking agent. PH is carcinogenic in mice following oral administration: 4/25 female and 3/25 male mice (of unspecified strain) developed thymic tumors within 450 days of feeding 1% PH in the diet; one thymus tumor was observed in 25 control males. The iv and oral LD50's of PH in mice are about 46 and 550 mg/kg, respectively. No data are available as to the carcinogenicity of PH in man. (11 refs.)

- 77-2415 **Formation and Metabolism of Alkylated Nucleosides: Possible Role in Carcinogenesis by Nitroso Compounds and Alkylating Agents.** (Eng.) Pegg, A. E. (Dept. Physiology, Milton S. Hershey Medical Center, Coll. Medicine, Pennsylvania State Univ., Hershey, PA) *Adv Cancer Res* 25: 199-269; 1977.

The formation and metabolism of both naturally occurring and carcinogen-produced alkylated nucleosides are reviewed. In contrast to the great specificity of the methylation of nucleic acids by physiological processes, the attack on nucleic acids by carcinogenic alkylating agents leads to the formation of alkylated nucleosides at many different sites throughout the cellular nucleic acids. Only a few of the alkylated products (7-methylguanine, 1-methyladenine, and 3-methylcytosine) have normal counterparts in cellular nucleic acids. The production of methylated nucleosides at regions within the nucleic acid sequence in which they are not normally found may have important biological consequences, such as inhibition of RNA and protein synthesis, misincorporation of incorrect

nucleosides by nucleic acid polymerases, stimulation of DNA repair processes, and, presumably, initiation of carcinogenesis. (414 refs.)

- 77-2416 **Nitrosamines--Environmental Carcinogens?** (Eng.) Walters, C. L. (British Food Manufacturing Industries Res. Assoc., Randalls Road, Leatherhead, Surrey KT22 7RY, England) *Chem Br* 13(4): 140-145; 1977.

Various precursors of N-nitroso compounds are found in the environment, along with bacteria that form the compounds, and catalysts of the formative process. Since it is impossible to judge the carcinogenic effect of natural exposures to trace amounts of these compounds, additional exposure to potentially dangerous compounds should be reduced. (31 refs.)

- 77-2417 **Inhaling Chemical Fumes Increases Risk of Cancers of Stomach, Esophagus, Larynx.** (Eng.) Anonymous (No affiliation given) *Public Health Rep* 92(3): 293; 1977.

The results of a preliminary biostatistical study on the risks of cancer associated with occupational exposure to chemical fumes and combustion products are summarized. Compared with persons inhaling combustion products, workers exposed to chemical fumes are at considerably greater risk of developing cancers of the stomach, the larynx, the mouth and pharynx, and the bladder. Relative risks for these cancers within a given occupation are cited according to age and length of time on the job. (no refs.)

- 77-2418 **Social and Ethical Implications of Claims for Cancer Hazards.** (Eng.) Weisburger, J. H. (Naylor Dana Inst. for Disease Prevention, American Health Foundation, Valhalla, NY 10595) *Med Pediatr Oncol* 3(2): 137-140; 1977.

Public statements have made claims for cancer hazards (eg, Red Dye #2, chlorinated hydrocarbons) that are not supported by epidemiologic and experimental evidence. Research funds diverted to investigate these claims dilute serious efforts to reduce the established risks of cigarette smoking and diet. These two main etiologic factors account for most cancers of the respiratory tract, digestive tract, and endocrine-sensitive and reproductive organs. (no refs.)

- 77-2419 **Explant Culture of Rat Colon: A Model System for Studying Metabolism of Chemical Carcinogens (Meeting Abstract).** (Eng.) Autrup, H. (Human Tissue



Studies Section, Experimental Pathology Branch, Carcinogenesis, NCI, Bethesda, MD 20014) Harris, C. C.; Stoner, G. D.; Fugaro, S. *In Vitro* 13(3): 192; 1977. (no refs.)

77-2420 **In Vitro Testing for Chemical Toxicity: Mammalian Target Cells (Meeting Abstract).** (Eng.) Waters, M. D. (Environmental Protection Agency, Research Triangle Park, NC 27711) Huisinck, J. L. *In Vitro* 13(3): 192; 1977. (no refs.)

77-2421 **Cancer Risk (Letter to Editor).** (Eng.) Peto, R. (Dept. Regius Professor Medicine, Univ. Oxford, Oxford, England) *New Sci* 73(1040): 480-481; 1977.

Exposure to carcinogens does not necessarily result in cancer. If two or more degenerative changes must occur in a single cell before it becomes malignant, then chance becomes a significant factor; the second degenerative change may or may not strike the same cell that underwent the first change. (no refs.)

77-2422 **Adjuvant Chemotherapy: Correct Advice? (Letter to Editor).** (Eng.) Louie, A. (NCI, Bethesda, MD 20014) Von Hoff, D. D.; Rozenzweig, M.; Muggia, F. *JAMA* 237(16): 1691; 1977.

Adjuvant chemotherapy following surgery for colonic carcinoma could be harmful and toxic. No clinical trials have provided sufficient data on improved survival to warrant this approach. (8 refs.)

77-2423 **Second Neoplasm--A Complication of Cancer Chemotherapy.** (Eng.) Chabner, B. A. (NCI, Bethesda, MD 20014) *N Engl J Med* 297(4): 213-215; 1977.

A study of 5,455 ovarian cancer patients revealed 13 patients who developed acute nonlymphocytic leukemia after taking alkylating agents for 3 to 90 mo. The median latency period was approx 3.5 yr. Other studies on the risk of chemotherapy, alone or in combination with irradiation, are reviewed. (10 refs.)

77-2424 **Reaction Selectivity and Molecular Association in Photochemical Reactions of Nucleic Acids and Their Constituents.** (Eng.) Elad, D. (Dept. Organic Chemistry, Weizmann Inst. Science, Rehovot, Israel) *Pure Appl Chem* 49(4): 503-509; 1977.

The light-induced free-radical reactions of purine and pyrimidine bases, nucleosides, and nucleotides with a variety of substrates were reviewed and found to be selective for the purines. The selectivity results from the suppression of pyrimidine reactivity due to the presence of the purines. This phenomenon probably results from base stacking. Other findings indicate that the reactivity of adenosine moieties in photochemical and free-radical reactions, involving the 8-position of the purine, is affected by the location of the sugar-phosphate linkage. This feature and intramolecular base stacking determine the degree of selectivity of these reactions for the appropriate base in systems containing adenine and uracil. The photoalkylation of DNA with alcohols or amines leads to selective modification of the purine moieties. Specific covalent cross-linking of RNase with its competitive inhibitors cytidine 2'(3'),5'-diphosphate and uridine 2'(3'),5'-diphosphate occurs upon irradiation in the presence of acetone as a photosensitizer. These observations emphasize the role that purines play in the photochemical transformations of nucleic acids, including cross-linking with proteins. (28 refs.)

77-2425 **Cancer-causing Radiation.** (Eng.) Ullrich, R. L. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Holland, J. M.; Storer, J. B. *Chemistry* 50(3): 7-11; 1977.

Mechanisms by which ionizing radiation can cause cancer are discussed, particularly the question of linear dose-response versus multiple factor analysis. The results of a few animal and human epidemiological studies are reviewed. (no refs.)

77-2426 **Radiation Exposure.** (Eng.) Anonymous (No affiliation given) *Arch Intern Med* 137(4): 417; 1977.

A review of 327 patients with chronic myelogenous leukemia indicated that almost 10% (29) had been exposed to radiation. The mean interval between exposure and diagnosis was 14.6 yr. (no refs.)

77-2427 **Radiation-Induced Health Effects (Letter to Editor).** (Eng.) Morgan, K. Z. (Sch. Nuclear Engineering, Georgia Inst. Technology, Atlanta, GA 30332) *Science* 195 (4276): 344, 346, 348; 1977.

The author supports an American Physical Society (APS) report that improves a previous one on radiation-induced health effects. The first report failed to take into account the long-term risks to the population and the seriousness of land

contamination from radioactive contaminants, especially cesium-137. It was also remiss in not including cancer and genetic deaths as one of the consequences of a reactor accident. There is strong evidence of an increased cancer incidence resulting from exposures to ionizing radiations equal to or less than those accepted as the max permissible levels for radiation workers. Two examples are given: (1) in 11,000 migrants into Israel to whom x-rays (6.5 rads) had been administered to control ringworm, there was a very high risk of thyroid carcinoma ( $6.1 \times 10^{-6}$  carcinomas/yr/rad administered); and (2) in children who received in utero radiation doses of 0.3-0.8 rad, mortality from leukemia and other forms of cancer was 50% higher than among unexposed controls. Children with diseases such as asthma, hives, eczema, allergy, pneumonia, dysentery, or rheumatic fever have a 5,000% greater risk of developing leukemia as a result of exposure to x-rays than children not exposed. The author also takes exception to the belief that there is a large factor of safety when applying the linear theory of radiation risk. There is strong theoretical and experimental evidence that the linear hypothesis, especially for high linear energy transfer radiation from neutrons and  $\alpha$  particles, underestimates the risk. (6 refs.)

- 77-2428 **Exposure Hazards of UV Radiation.** (Eng.) Hughes, D. (Univ. Leeds, Leeds LS2 9JT, England) *Chem Br* 13(4): 134-137; 1977.

Animal studies have indicated that exposure to UV radiation of wavelengths below 320 nanometers is carcinogenic. Furthermore, numerous skin cancers have been diagnosed in workers exposed for long periods to direct solar radiation. Various exposure rates and threshold values are listed. (25 refs.)

- 77-2429 **The Distrust of Nuclear Power.** (Eng.) Hohenemser, C. (Dept. Physics, Clark Univ., Worcester, MA 01610) Kasperson, R.; Kates, R. *Science* 196(4285): 25-34; 1977.

The accident risk per yr in deaths per million for 100 nuclear reactors in the United States would range from 0.02 to 0.2, taking into account the possibility of delayed cancer deaths. Furthermore, although plutonium is a potent carcinogen, no cancers can definitely be attributed to exposure among the thousands of workers handling it. (77 refs.)

- 77-2430 **Cosmetic Talc Powder.** (Eng.) Anonymous (No affiliation given) *Lancet* 1(8026): 1348-1349; 1977.

Various epidemiological studies on the effects of cosmetic talc powder are reviewed. It does not appear that exposure to talc leads to loss of lung function or to cancer of any type. (18 refs.)

- 77-2431 **Carcinomatous Change in Old Scars.** (Eng.) Kennedy, T. J. (Milton S. Hershey Medical Center, Hershey, PA) Miller, S. H.; Graham, W. P.; Davis, T. S. *Am Fam Physician* 16(1): 106-107; 1977.

Malignant transformation in the scar tissue of a burn is usually manifest 20 or 30 yr after the original injury. Infection or delayed healing and continued trauma to the scar after healing are necessary conditions for malignant development over this extended time period. (no refs.)

- 77-2432 **Biological Activity of Tumor Virus DNA.** (Eng.) Graham, F. L. (Dept. Biology, McMaster Univ., Hamilton, Ontario, Canada) *Adv Cancer Res* 25: 1-51; 1977.

There are several advantages to using purified viral DNA to infect cells as opposed to using intact virions: (1) it is possible to modify the DNA by physical, chemical, and biochemical means and (2) it may be possible to infect with DNA cells that are resistant to infection by intact virions as a result of blocks in adsorption, uptake, or uncoating. An extensive review is presented of the development and use of the more common techniques for assaying the biological activity of purified viral DNA and of some of the studies in which these assays have been used to probe the structure and function of tumor virus DNA. Transformation studies have led to the conclusion that fragments of viral DNA of less than genome size can transform cultured cells and induce tumors in animals. This transformation by subgenomic fragments of DNA from simian virus 40 or from adenovirus has been clearly demonstrated, and preliminary studies suggest that transformation may also be possible with fragments of herpes simplex virus DNA. (215 refs.)

- 77-2433 **How Do Tumour Viruses Transform Cells? DNA Tumour Viruses.** (Eng.) Sambrook, J. (Cold Spring Harbor Lab., Cold Spring Harbor, NY) *Trends Biochem Sci* 2(7): 152-154; 1977.

The transformation of nonpermissive cells by the DNA papovaviruses polyoma and SV40 probably occurs by the integration of the viral protein into the A gene protein of the host cell. The crucial role of the T antigen in the transformation process is discussed. (22 refs.)



- 77-2434 **Terminal Redundancy in Avian Oncornavirus RNA.** (Eng.) Harris, T. J. (Biochemistry Dept., Animal Virus Res. Inst., Pirbright, Surrey, England) *Nature* 268(5615): 15; 1977.

The avian oncornavirus employs reverse transcriptase to make a DNA copy of itself upon infection of cells. Evidence now indicates that both the 3' and 5' ends of the virus are complementary, allowing both the virus RNA and the complementary DNA to form circles. (6 refs.)

- 77-2435 **Mouse "Tumour Gene" is a Disappointment.** (Eng.) Anonymous (No affiliation given) *New Sci* 73(1035): 141; 1977.

Experimental attempts at finding an oncogene in mice are reviewed. DNA was matched to the "sarc" gene (the gene necessary for the transforming properties of the tumor virus). Probes of normal and transformed RNA failed to uncover the oncogene sarc sequences. (no refs.)

- 77-2436 **Malignancy and Transformation: Expression in Somatic Cell Hybrids and Variants.** (Eng.) Ozer, H. L. (Worcester Foundation for Experimental Biology, Shrewsbury, MA) Jha, K. K. *Adv Cancer Res* 25: 53-93; 1977.

Current data regarding the expression of the transformed state in somatic cell hybrids are summarized. Suppression of malignancy can occur in intraspecies cell hybrids between malignant and nonmalignant cells and with a large variety of tumors of different origin. Similarly, a variety of "normal" cells are capable of suppression: lymphocytes, fibroblasts, and even cells passaged extensively in vitro, such as L cells, although some sublines and individual cells in most sublines are themselves capable of tumor formation. A tentative conclusion from cell fusion studies is that only a limited number of mutations can result in the establishment of a neoplasm. This impression is supported by the limited degree of "complementation" observed in cell hybrids between malignant cell lines. Discussion is made of the isolation of cell hybrids, the expression of malignancy in cell hybrids (malignant x intraspecies nonmalignant cells, malignant x interspecies nonmalignant cells, and malignant x malignant cells), and the expression of the transformed phenotype in vitro by cell hybrids. (150 refs.)

- 77-2437 **Viruses and Breast Cancer: A Prospective View of Approaches Applicable to Primary Prevention and Early Detection of Human Breast Cancer.** (Eng.) Schlom, J. (Viral Oncology Branch, NCI, Bethesda, MD 20014) *Cancer Detect Prevent* 1(2): 255-261; 1976.

Although there is immunological and biochemical evidence for the presence of RNA tumor viruses in human breast cancer, there is as yet no overwhelming evidence that any particular virus or viruses are etiologically involved in the disease. Experimental studies of virally associated breast cancers in animals, the prospect of an antiviral vaccine for human breast cancer, and the possible early detection of human breast cancer by identification of viral antigens are discussed. (14 refs.)

- 77-2438 **Australia Antigen and the Biology of Hepatitis B.** (Eng.) Blumberg, B. S. (Inst. Cancer Res., Fox Chase Center, Philadelphia, PA 19111) *Science* 197(4298): 17-25; 1977.

Genetic, immunological and viral factors involved in the susceptibility to and spread of hepatitis B infection are reviewed. The relation of hepatitis B to primary hepatic carcinoma has been established using the Australia antigen test. In some cases children infected with viral hepatitis by the mother before, at, or shortly after birth may proceed through several stages leading to the development of primary hepatic carcinoma. (54 refs.)

- 77-2439 **Medical Complications of Renal Transplantation. Part II. Noninfectious Complications in Recipient.** (Eng.) Prompt, C. A. (Div. Nephrology, UCLA, Medical Center, Los Angeles, CA 90024) Lee, D. B.; Upham, A. T.; Kleeman, C. R. *Urology (Suppl)* 9(6): 32-48; 1977.

Documented noninfectious complications in kidney transplant patients are reviewed, including de novo neoplasms. Of 256 lesions identified by a Denver tumor registry for transplant patients, 39% were cancers of the skin and lips; other tumors included solid lymphomas (27%), carcinomas of the cervix (7%), and carcinomas of the lungs (5%). Data suggest that immunosuppression may facilitate the development of neoplasias. (178 refs.)

- 77-2440 **Immunosuppression and the Role of Suppressive Factors in Cancer.** (Eng.) Kamo, I. (Dept. Microbiology and Immunology, Albert Einstein Medical Center, Philadelphia, PA) Friedman, H. *Adv Cancer Res* 25: 271-321; 1977.

The role of immunosuppressive factors in cancer is reviewed. Evidence that a number of divergent mechanisms, either separately or in various combinations, may subvert the normal immune defense mechanism of an individual against tumor cells is discussed. Discussion is made of (1) general aspects and the mechanism of immune suppression in tumor-

bearing individuals (T-cell immunodeficiencies, tumor effects on lymphocyte trapping, depressed phagocytic activity, altered B-cell number and activity, the role of "suppressor" cells in malignancies, and immunological memory in tumor-bearing individuals) and (2) immunosuppressive humoral factors during malignancies (blocking factors, tumor and fetal tissue antigens, normal immunoregulatory proteins, low-molecular-wt suppressive factors and immunosuppressive tumor cell extracts, ascites fluids, and plasma). (355 refs.)

- 77-2441 **Tumor-Bound Immunoglobulins: In Situ Expressions of Humoral Immunity.** (Eng.) Witz, I. P. (Dept. Microbiology, Dr. George S. Wise Center Life Sciences, Tel Aviv Univ., Tel Aviv, Israel) *Adv Cancer Res* 25: 95-148; 1977.

A comprehensive review is presented of the presence, properties, and functions of humoral components, mainly immunoglobulins (Ig), at the site of malignant tumors. The topics covered include properties of the tumor-associated immunoglobulins (TAI), ie, the Ig class, the IgG subclass, changes in the level of TAI in transplanted tumors with time after implantation, and the dynamic state of TAI; the nature of Ig-associated cells in tumors; antitumor antibodies; the presence of receptors for immune complexes within tumors; the degradation of antitumor antibodies; and biological functions of TAI. (300 refs.)

- 77-2442 **Transfer Factor: Is It Related to Immune RNA?** (Eng.) Valentine, F. T. In: *Immune RNA in Neoplasia*. Fink, M. A., ed. (New York: Academic Press, Inc.): pp. 75-84; 1976.

Intriguing problems involved with the molecular identification of transfer factor (TF) and the possible relationship of TF to immune RNA are discussed. Biological and immunological observations concerning TF are described. Several lines of evidence, including that provided by studies on the transfer of the accelerated homograft rejection, suggest that TF transfers immunologically specific cell-mediated immunity to nonsensitive humans. When patients afflicted with osteogenic sarcoma were injected with TF from donors sensitive to the tumor, recipient lymphocytes temporarily acquired the ability to destroy this tumor in vitro. The results of several observations suggest that antigen is not present in TF. The resistance of TF to pancreatic RNase is unlike the characteristics of immune RNA, although the dialyzable active component may consist in part of protected RNA. In patients with diseases associated with immunological defi-

encies of unknown cause, TF had immunologically non-specific effects. The possible role of TF in treating human disease is being investigated. (39 refs.)

- 77-2443 **Germ Cell Tumors of the Testes.** (Eng.) Braunstein, G. D. (Dept. Medicine, Cedars-Sinai Medical Center, 8720 Beverly Blvd. No. 6736, Los Angeles, CA 90048) Friedman, N. B.; Sacks, S. A.; Skinner, D. G.; Tompson, R. W. *West J Med* 126(5): 362-377; 1977.

The classification, pathogenesis, and treatment of germ cell tumors of the testes are discussed. The tumors are divided into five basic types: seminomas, embryonal carcinomas, teratocarcinomas, adult teratomas, and choriocarcinomas. Clinically, they may present as an enlarging testicular mass, or with symptoms resulting from metastases or hormonal secretions. The treatment of choice for patients with seminomas is orchiectomy, followed by radiation therapy; this combination results in an 80%-100% 5-yr survival rate in patients with nonmetastatic or locally metastatic disease. The treatment of nonseminomatous germ cell tumors is more controversial, but retroperitoneal lymph node dissection and adjuvant chemotherapy are associated with an overall 78% survival rate. Of the placental and fetal proteins secreted by these neoplasms, human chorionic gonadotropin and  $\alpha$ -fetoprotein are useful for tumor diagnosis, for following the response to therapy, and for detection of recurrences. (134 refs.)

- 77-2444 **Tumor Cell Surfaces: Some Characteristics of Neoplastic Cells That Determine States of Transformation and Malignancy.** (Eng.) Nicolson, G. L. (Dept. Developmental and Cell Biology, Univ. California, Irvine, CA 92717) Brunson, K. W.; Fidler, I. J. *Acta Histochem Cytochem* 10(1): 114-133; 1977.

Normal and neoplastic cells are surrounded by fluid, dynamic plasma membranes that are involved in a variety of important physiological processes. Although several modifications in composition, organization, dynamics, enzymology, and immunology have been found in transformed and tumor cell membranes, compared with their untransformed and normal cell counterparts, few of these changes may be important in the actual survival, growth and spread of tumor cells in vivo. One of the most important characteristics of malignant cells in vivo--the ability to metastasize to distant host sites--provides an important approach to studying in vivo tumor cell properties. Selection procedures are described that were used to obtain variant murine B16 melanoma cell lines with enhanced metastatic potential and ability to spread to specific host sites (lungs, brain) by bloodborne routes. The use of these selected variants may provide insight into the cell. (114 refs.)



## CHEMICAL CARCINOGENESIS

### 77-2445 Investigations of Cigarette Smoke Dosages in Inhalation Experiments with Syrian Hamsters.

I. Concentration of Cigarette Smoke in the Inhalation Chamber and of Carbon Monoxide in the Blood. (Eng.) Klimisch, H. J. (BASF Aktiengesellschaft-WOT-J 560, D-6700 Ludwigshafen, Rhein, W. Germany) Döntenwill, W. *J Natl Cancer Inst* 58(4): 931-933; 1977.

Tests performed on 20 different smoke inhalation machines demonstrated that 60% of the gas phase and 40% of the total particulate matter reached the inhalation chamber with the remainder lost in the exhaust gases. Based on these percentages, CO and CO<sub>2</sub> concentrations were calculated for each puff of smoke administered to Syrian hamsters in the inhalation chamber. CO-Hb levels in 10 hamsters exposed for 20 min to the smoke from 30 cigarettes with a 4.3% CO content rose to 61.5%. The limiting factor in inhalation experiments is the toxic response to nicotine or CO. It is concluded that the smoke exposure time should not exceed 20 min and the max allowable CO-Hb level should be 60%. (4 refs.)

### 77-2446 Cigarette Smoking and Cancer of Bladder and Lung (Letter to Editor). (Eng.) Edwards, T. A. (Chest Clinic, St. Albans City Hosp., St. Albans, Herts, England) *Br Med J* 1(6061): 637-638; 1977.

Case reports are presented for two men, both smokers, who developed bladder cancer subsequent to the development of lung cancer. The bladder cancers were diagnosed 10 to 14 yr after surgical intervention for the lung cancer, possibly indicating a longer latent period. (no refs.)

### 77-2447 Fractionation of Mouse Skin Carcinogens in Cigarette Smoke Condensate. (Eng.) Lee, P. N. (Tobacco Res. Council, Glen House, Stag Place, London SW1E 5AG, England) Rothwell, K.; Whitehead, J. K. *Br J Cancer* 35(6): 730-742; 1977.

The results of a series of mouse-skin paintings are given for fractions prepared by two techniques designed to concentrate the polycyclic aromatic hydrocarbons (PAH) and their heterocyclic analogs (HETC) in cigarette smoke condensates into single fractions. For each group, a single index of tumor response, the Weibull risk parameter (WRP), was calculated

that, considered in conjunction with two other parameters common to all the groups, adequately described the pattern of tumor incidence in that group. These indices were used to calculate a further statistic for each fraction, the tumorigenic ratio (TR), which measures the activity of the fraction relative to whole-smoke condensate on a wt-for-wt basis. The test results show that the separation processes can successfully concentrate all types of mouse-skin carcinogenic material, irrespective of the type of condensate used, and that a combination of processes prepares an active concentrate representing 2% by wt of the original condensate. (10 refs.)

### 77-2448 The Effects of Cigarette Smoke on the Metabolism of [<sup>3</sup>H]Benzo(a)pyrene by Rat Lung Microsomes. (Eng.) Uotila, P. (Dept. Physiology, Univ. Turku, FIN-20520, Turku 52, Finland) Pelkonen, O.; Cohen, G. M. *Cancer Res* 37(7): 2156-2161; 1977.

The metabolism of <sup>3</sup>H-benzo(a)pyrene (BP: 0.5 μM) and the activities of specific enzymes involved in its further metabolism were studied in lung microsomes from sham- and cigarette smoke-exposed and 3-methylcholanthrene (MC)-pretreated (20 mg/kg ip for 3 days) rats. BP was converted into metabolites cochromatographing with reference dihydrodiols, phenols, and quinones as well as some unknown metabolites. Exposure of rats to cigarette smoke increased the formation of different metabolites from three- to sixfold, whether the exposure was for 1, 10, or 21 days. The metabolite patterns were similar in smoke-exposed and MC-pretreated rats, but MC caused a greater increase in metabolite production. Pulmonary aryl hydrocarbon hydroxylase activity increased from three- to sixfold after smoke exposure. The activity of epoxide hydratase (substrate: styrene oxide) decreased after 1 day of smoke exposure and did not change after exposure for 10 or 21 days. Glutathione S-transferase activity (substrate: styrene oxide) increased after 1 and 10 days of smoke exposure. No significant changes could be seen in the activity of uridine diphosphate glucuronosyltransferase (substrate, 4-methylumbelliferone). Isolated perfused rat lungs and lung microsomes converted BP to similar metabolites, but differences were observed in both the absolute and relative amounts of the different metabolites. 9,10-Dihydro-9,10-dihydroxybenzo(a)pyrene and 7,8-dihydro-7,8-dihydroxybenzo(a)pyrene were the major dihydrodiols formed in perfusion experiments, but 4,5-dihydro-4,5-dihydroxybenzo(a)pyrene was the major metabolite in the microsomes. This difference was probably due to differing rates of further conjugation of primary metabolites in the isolated perfused lungs. (49 refs.)

- 77-2449    **The Phototoxic Activity of Carcinogenic Polycyclic Hydrocarbons and Their Degradation Products in the Biological Test.** (Ger.) Graf, W. (Institut für Umwelthygiene und Präventivmedizin, Wasserturmstr. 5, D-8520 Erlangen, W. Germany) Haller, A. G. *Zentralbl Bakteriol [Orig B]* 164(3): 250-256; 1977.

The phototoxic activity of carcinogenic polycyclic hydrocarbons was evaluated using ciliates of *Tetrahymena pyriformis* maintained in sterile, semisynthetic media. This ciliate light test was developed into and standardized for a rapid microbiological assay of carcinogenic activity of polycyclic aromatic hydrocarbons. Six compounds with differing carcinogenic activities were tested: benzo(a)pyrene (BP), benzo(b)fluoranthene, indeno(1,2,3-c,d)pyrene, benzo(a)anthracene, fluoranthene, and cyclopenteno(c,d)pyrene. An essential parallelism between their phototoxic and carcinogenic activities was obvious. The combined effects of BP and fluoride ions were also investigated using the ciliate light test. Fluoride ions (1 ppm) did not affect the phototoxicity of BP, suggesting that fluorides possess no cocarcinogenic activities. Degradation products of BP obtained by UV irradiation (mixtures of quinones) were also tested for their phototoxic activity. These degradation products, entirely free of BP, still possessed 50% of the activity of the original, nonirradiated solution. This indicates that the degradation products resulting from UV irradiation may still retain a measure of carcinogenic activity, a finding of considerable importance from the environmental viewpoint. (9 refs.)

- 77-2450    **Relationship Between Structure and Reactivity of Benzo(a)pyrene and Several Methylated Derivatives.** (Fre.) Paalme, L. (Institut de Chimie de l'Académie des Sciences de la République Socialiste Soviétique d'Estonie, 21, Akademies tee Tallinn, USSR) Perin-Roussel, O.; Goubergrits, M.; Jacquignon, P. *J Chim Phys* 74(4): 496-498; 1977.

An attempt was made to correlate structure and reactivity of methylated derivatives of benzo(a)pyrene during photodegradation. The degradation of these compounds was fastest in the presence of oxygen; the reactivities are presented. (7 refs.)

- 77-2451    **Stereochemistry of the Hydrolysis Products and Their Acetonides of Two Stereoisomeric Benzo[a]pyrene 7,8-Diol 9,10-Epoxides.** (Eng.) Yang, S. K. (Chemistry Branch, NCI, NIH, Bethesda, MD 20014) McCourt, D. W.; Gelboin, H. V.; Miller, J. R.; Roller, P. P. *J Am Chem Soc* 99(15): 5124-5130; 1977.

The stereochemistry of r-7, t-8-dihydroxy-t-9, 10-oxy-7,8,9,10-tetrahydrobenzo[a]pyrene and r-7,t-8-dihydroxy-c-

9,10-oxy-7,8,9,10-tetrahydroxybenzo(a)pyrene, metabolites possibly responsible for the carcinogenicity of benzo[a]pyrene, was established by ultraviolet absorption spectra, reactions with potassium triacetylosmate, and mass spectra analysis following high-pressure liquid chromatography. The results indicated that carbonium ions formed at C(10) are the intermediates in the hydrolysis of both diol epoxides. (36 refs.)

- 77-2452    **Organic Solvent Soluble Sulphate Ester Conjugates of Monohydroxybenzo(a)pyrenes.** (Eng.) Cohen, G. M. (Dept. Biochemistry, Univ. Surrey, Guildford, GU2 5XH, Surrey, England) Moore, B. P.; Bridges, J. W. *Biochem Pharmacol* 26(6): 551-553; 1977.

The production of acetate-soluble conjugates of both 7-hydroxybenzo(a)pyrene and 9-hydroxybenzo(a)pyrene is reported. The biological importance of these conjugates and their metabolites remains to be determined. (13 refs.)

- 77-2453    **Analysis of the Biotransformation of Benzo(a)pyrene in Human Fetal and Placental Tissues with High-Pressure Liquid Chromatography.** (Eng.) Berry, D. L. (Oak Ridge Natl. Labs., Oak Ridge, TN 38730) Zachariah, P. K.; Slaga, T. J.; Juchau, M. R. *Eur J Cancer* 13(7): 667-673; 1977.

The biotransformation of benzo(a)pyrene (BP) was studied in vitro in the presence of microsomal fractions of placental and fetal tissues from humans and monkeys (*Macaca nemestrina*). Metabolites formed in the incubation flasks were extracted and separated by high-pressure liquid chromatography utilizing a microparticulate column. In general, the formation of diols and quinones in fetal and placental homogenates was undetectable following 15-min incubations. The formation of phenolic metabolites, however, was easily measurable in fetal liver and lung and in the placenta, but not in fetal spleen, kidney, pancreas, or adrenal gland. The latter observation contrasted with the high specific activities measured in the fetal adrenal gland by a fluorometric assay for aryl hydrocarbon hydroxylase activity. In placentas from cigarette smokers, relatively large quantities of an unidentified metabolite(s) appeared in the metabolic profiles. This metabolite(s) did not cochromatograph with any of the standard metabolites, and the retention time was between those of the 9,10- and 4,5-diols. The same placental tissues catalyzed the covalent binding of BP to DNA, and they were far more active in this regard than any of the other fetal tissues investigated. The data indicated a correlation between metabolic profiles and capacity for catalyzing covalent binding to DNA for fetal placental tissues. (33 refs.)



- 77-2454 **Microsomal Metabolism of Benzo(a)pyrene: Multiple Effects of Epoxide Hydrase Inhibitors.** (Eng.) Fahl, W. E. (Dept. Pharmacology, Univ. Wisconsin Medical Sch., Madison, WI 53706) Nesnow, S.; Jefcoate, C. R. *Arch Biochem Biophys* 181(2): 649-664; 1977.

The relationship between polycyclic hydrocarbon oxidation and arene oxide hydrase (AO hydrase) was examined by studying the metabolism of benzo(a)pyrene (BP) by rat liver microsomes in the presence of competitive (styrene oxide), uncompetitive (3,3,3-trichloropropene oxide, TCPO), and noncompetitive (cyclohexene oxide) inhibitors of AO hydrase. Formation of BP dihydrodiols was inhibited selectively in 3-methylcholanthrene (MC)-induced and phenobarbital (PB)-induced microsomes. The 9,10-dihydrodiol was most sensitive to the inhibitors, followed by the 7,8-dihydrodiol and the 4,5-dihydrodiol. Increased levels of 9-phenol, 7-phenol, and 4,5-oxide appeared selectively in the same order. The appearance of these alternate products did not compensate quantitatively for the loss of dihydrodiols, so that there was a net loss of oxidation products. The limiting decrease in BP oxidation products was the same for each inhibitor, and it was greater for MC-induced microsomes (25%-30%) than for PB-induced microsomes (15%-20%). A complete kinetic description of the microsomal metabolism of BP is very complex, but many features of the process can be described by the use of first-order kinetics for the enzymatic hydration of BP oxides. (43 refs.)

- 77-2455 **Factors Influencing Aryl Hydrocarbon Hydroxylase (AHH) Activity in Human Lymphocytes** (Meeting Abstract). (Eng.) Kouri, R. E. (Dept. Biochemical Oncology, Microbiological Associates, Bethesda, MD 20016) McKinney, C.; Sosnowski, R.; Schechtman, L. M. *Proc Am Assoc Cancer Res* 18: 150; 1977. (no refs.)

- 77-2456 **Aryl Hydrocarbon Hydroxylase in Mouse Mammary Gland: In Vitro Study Using Mammary Cell Lines.** (Eng.) Chuang, A. H. (Dept. Biochemistry, Univ. Vermont Coll. Medicine, Burlington, VT 05401) Howard, E. F.; Bresnick, E. *Chem Biol Interact* 17(1): 9-16; 1977.

The effects of 3-methylcholanthrene (MC), 5,6-benzoflavone ( $\beta$ NF), 7,8-benzoflavone ( $\alpha$ NF), and pregnenolone 16 $\alpha$ -carbonitrile (PCN) (at concentrations of  $10^{-5}$ M) on aryl hydrocarbon hydroxylase (AHH) levels were determined in primary mammary gland epithelial cell cultures prepared from C3Hf/Ki mice. MC elevated AHH activity three- to fourfold after 24 hr of treatment,  $\beta$ NF and PCN increased the activity by 86% and 33%, respectively, but  $\alpha$ NF produced a 50% inhibition. The specific activity of AHH in these cells was elevated by 6 hr after exposure to MC; enzyme activity was still max elevated after 48 hr. The effects of MC were also

investigated in several mammary cell lines: one derived from a control virgin mouse, MCG V14; three from mammary tumors, MCG T10, MCG T14, and MCG T19; and two from hyperplastic alveolar nodules, HAN-1 and HAN-2. Induction was seen in all lines at 24 hr with MCG T14 the most responsive and HAN-2 the least. Although the MCG T19 tumor cells responded in culture, the AHH of the tumors induced by the cells in recipient A+/Ki was not elevated by administration of MC (80 mg/kg ip) in vivo. (27 refs.)

- 77-2457 **Effect of Inducers and Inhibitors of Mixed-Function Oxidases on the In Vivo Metabolism of Dioxane in Rats** (Meeting Abstract). (Eng.) Woo, Y. T. (USPHS Res. Lab., Tulane Medical Center, 210 State St., New Orleans, LA 70118) Argus, M. F.; Arcos, J. C. *Proc Am Assoc Cancer Res* 18: 165; 1977. (no refs.)

- 77-2458 **Aryl Hydrocarbon Hydroxylase Activity in Tracheal Epithelium of Syrian Golden Hamsters** (Meeting Abstract). (Eng.) Mass, M. J. (Dept. Pathology, Univ. North Carolina, Chapel Hill, NC 27514) Kaufman, D. G. *Proc Am Assoc Cancer Res* 18: 142; 1977. (no refs.)

- 77-2459 **Measurement and Reproducibility of Aryl Hydrocarbon Hydroxylase (AHH) in Cultured Human Lymphocytes** (Meeting Abstract). (Eng.) Gurtoo, H. L. (Roswell Park Memorial Inst., Buffalo, NY 14263) Minowada, J.; Parker, N. B.; Hayner, N. T. *Proc Am Assoc Cancer Res* 18: 55; 1977. (no refs.)

- 77-2460 **Michaelis-Menten Kinetic Analysis of Aldrin Epoxidase on Microsomes and Isolated Rat Hepatocytes** (Meeting Abstract). (Eng.) Dubois-Krack, G. (Laboratoire de Biotoxicologie, Ecole de Pharmacie, Université de Louvain, 1200 Bruxelles, Belgium) Roberfroid, M.; Bettencourt, A.; Mercier, M. *Arch Int Physiol Biochim* 85(2): 409-410; 1977. (2 refs.)

- 77-2461 **Induction of Cholesteatoma in Experimental Animals** (Meeting Abstract). (Ger.) Steinbach, E. (Tubingen, W. Germany) *Zentralbl Allg Pathol* 121(3): 293; 1977. (no refs.)

77-2462 **Variability of Physiological and Karyotypic Characteristics of BALB/c-3T3 Cell Populations (Meeting Abstract).** (Eng.) Sivak, A. (Arthur D. Little, Inc., Cambridge, MA 02140) Rudenko, L.; Simons, I. *In Vitro* 13(3): 198; 1977. (no refs.)

77-2463 **In Vitro Metabolic Activation Systems (Meeting Abstract).** (Eng.) Kouri, R. E. (Dept. Biochemical Oncology, Microbiological Assoc., Bethesda, MD 20014) Schechtman, L. M. *In Vitro* 13(3): 192; 1977. (no refs.)

77-2464 **Binding of Benzo[a]pyrene at 1, 3 and 6 Positions to Nucleic Acids In Vitro and In Vivo (Meeting Abstract).** (Eng.) Rogan, E. (Eppley Inst., Univ. Nebraska Medical Center, Omaha, NB 68105) Roth, R.; Cavalieri, E.; Katomski, P.; Benderson, J. *Proc Am Assoc Cancer Res* 18: 59; 1977. (no refs.)

77-2465 **High Carcinogenicity of 2-Hydroxybenzo(a)pyrene (2-HOBP) on Mouse Skin (Meeting Abstract).** (Eng.) Wislocki, P. G. (Hoffmann-La Roche Inc., Nutley, NJ 07110) Kapitulnik, J.; Levin, W.; Yagi, H.; Dansette, P. M.; Jerina, D. M.; Conney, A. H. *Proc Am Assoc Cancer Res* 18: 140; 1977. (1 ref.)

77-2466 **Binding of Benzo(a)pyrene-Semiquinone Radicals with DNA and Polynucleotides.** (Eng.) Kodama, M. (Biophysics Div., Natl. Cancer Center Res. Inst., Tsukiji 5-1-1, Chuo-ku, Tokyo 104, Japan) Ioki, Y.; Nagata, C. *Gann* 68(2): 253-254; 1977.

The binding between the benzo(a)pyrene (BP)-semiquinone radical and DNA or polynucleotides was investigated. Aqueous solutions of calf thymus DNA were added to solutions of semiquinone radicals, and incubated at 37 C for 18 hr before being prepared for electron-spin resonance (ESR) measurement. The ESR signals indicated that binding of the 3,6-semiquinone radical was largest, twice that of the 1,6-semiquinone radical. No binding could be detected when 6,12-semiquinone was reacted with DNA. Base specificity in the binding was investigated by using poly(G), poly(A), poly(C), and poly(U). The ESR signal due to the bound complex was extremely large for poly(G), but the other polynucleotides gave signals that were only 5% that for poly(G), indicating that the guanine residue is the binding site. Analysis of the g and line width values, which characterize the structure

of the free radical, suggest that semiquinone radicals are active species in binding to the base moiety. (5 refs.)

77-2467 **Effects of Butylated Hydroxyanisole (BHA) on the Metabolism of Benzo(a)pyrene (BP) by Mouse Liver Microsomes (Meeting Abstract).** (Eng.) Lam, L. K. (Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN 55455) Wattenberg, L. W. *Proc Am Assoc Cancer Res* 18: 163; 1977. (no refs.)

77-2468 **Enhancement of Mutagenesis In Vitro by a Tumor Promoting Agent (Meeting Abstract).** (Eng.) Lankas, G. R. (Kettering Lab., Univ. Cincinnati, Cincinnati, OH 45267) Christian, R. T.; Baxter, C. S. *In Vitro* 13(3): 193-194; 1977. (no refs.)

77-2469 **Vitamin A Acid (Retinoic Acid), a Potent Inhibitor of 12-O-Tetradecanoyl-phorbol-13-acetate-induced Ornithine Decarboxylase Activity in Mouse Epidermis.** (Eng.) Verma, A. K. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI 53706) Boutwell, R. K. *Cancer Res* 37(7): 2196-2201; 1977.

Topical application of retinoic acid to Charles River mouse skin led to a dramatic inhibition of 12-O-tetradecanoyl phorbol-13-acetate (TPA)-induced epidermal ornithine decarboxylase (OD) activity, an event proposed to be essential for tumor promotion. The degree of inhibition was dependent on the dose and time of application of retinoic acid; enzyme activity was depressed 90% or more when 1.7 nanomoles (nmol) of retinoic acid was applied 1 hr before TPA at 17 nmol. In contrast, treatment with retinoic acid did not depress significantly TPA-induced S-adenosylmethionine decarboxylase activity, a second enzyme in the pathway of polyamine biosynthesis. A number of natural vitamin A analogs (retinoids) were tested for their ability to inhibit TPA-induced epidermal OD activity. They were found to be potent in the following order: retinoic acid > retinal > retinol > retinyl acetate > retinyl palmitate. The ability of retinoids to inhibit TPA-induced epidermal OD activity correlated with their ability to inhibit skin tumor promotion. Mixing of soluble extracts from TPA-treated mouse epidermis pretreated with either retinoic acid or acetone gave essentially additive OD activity, arguing against the production of an inhibitor of TPA-induced OD activity. Furthermore, retinoic acid did not alter TPA-induced OD activity when added to the assay mixture under normal assay conditions. (38 refs.)

77-2470 **On the Carcinogenicity of 5-Methylchrysene (Meeting Abstract).** (Eng.) Hecht, S. S. (Naylor



Dana Inst. for Disease Prevention, American Health Foundation, Valhalla, NY 10595) Loy, M.; Mazzaresse, R.; Hoffmann, D. *Proc Am Assoc Cancer Res* 18: 120; 1977. (no refs.)

- 77-2471 **Heteroploid Conversion of Human Skin Cells by Methylcholanthrene.** (Eng.) Freeman, A. E. (Microbiological Associates, Torrey Pines Res. Center, 2945 Science Park Road, La Jolla, Ca 92037) Lake, R. S.; Igel, H. J.; Gernand, L.; Pezzutti, M. R.; Malone, J. M.; Mark, C.; Benedict, W. F. *Proc Natl Acad Sci USA* 74(6): 2451-2455; 1977.

Factors influencing the chemically induced transformation of human skin cells were investigated. Cultured human epithelial cells generally had three- to thirtyfold more hydrocarbon-metabolizing activity (HMA) than fibroblasts from the skin of the same donor. This activity was constant for up to 55 days in primary culture but was lost rapidly upon physical subdivision of the cultures. Treatment of primary mixed fibroblasts and epithelial cell cultures with methylcholanthrene, but not phenanthrene, led to the development of actively growing fibroblastic cultures with many heteroploid cells. Unique marker chromosomes, stable over a number of cell population doublings, were identified in several heteroploid cell strains. Pure cultures of fibroblasts from the same donors did not undergo heteroploid conversion in response to MC. Spontaneously occurring heteroploidy in logarithmic phase human fibroblasts is a rare event; thus, heteroploid conversion may be a useful marker for chemical transformation of human cells. Because of their higher levels of HMA, epithelial cells may convert MC to an ultimate carcinogenic form that causes heteroploidy in cocultured fibroblasts. (26 refs.)

- 77-2472 **Susceptibility of C3HeB/FeJ Mice to the Induction of Lung Carcinoma (Meeting Abstract).** (Eng.) Furst, A. (Inst. Chemical Biology, Univ. San Francisco, San Francisco, CA 94117) Kolff, K. B.; Ho, W. *Proc Am Assoc Cancer Res* 18: 138; 1977. (1 ref.)

- 77-2473 **3,4,3',4'-Tetrachloroazobenzene: A Potential Environmental Toxicant.** (Eng.) Hsia, M. T. (Dept. Pathology, Medical Sch., Univ. Wisconsin, Madison, WI 53706) Bairstow, F. V.; Shih, L. C.; Pounds, J. G.; Allen, J. R. *Res Commun Chem Pathol Pharmacol* 17(2): 225-236; 1977.

The in vitro toxicity of 3,4,3',4'-tetrachloroazobenzene (TCAB) was examined in C3H/10T1/2 mouse embryo fibroblasts. Growth inhibition of the fibroblasts was observed after 3 days of treatment with  $>2 \mu\text{g/ml}$  TCAB. After a 7-day incubation,  $0.78 \mu\text{g/ml}$  TCAB was also toxic to fibro-

blasts. Within 24 hr of exposure to TCAB, all cells contained numerous, highly refractile structures, and the adjacent cytoplasm was coarse compared to control cells. These cytological changes were reversible when the TCAB medium was replaced with fresh medium. TCAB was weakly mutagenic by the Salmonella/mammalian-microsome test. In addition, C3H/10T1/2 cells exposed to TCAB for 10 days showed focal morphologic alterations characteristic of transformed cells. The rapid appearance of these foci suggests that TCAB is oncogenic, but more definite evaluation is required. These data suggest that there is a potential health hazard associated with the environmental presence of TCAB. (14 refs.)

- 77-2474 **Evidence for a Receptor Protein of Activated Carcinogen.** (Eng.) Mainigi, K. D. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA 19111) Sorof, S. *Proc Natl Acad Sci USA* 74(6):2293-2296; 1977.

The principal carcinogen-protein complex isolated from the livers of male CD rats fed 3'-methyl-4-dimethylaminoazobenzene (3'-MeDAB, 580 mg/kg diet) for up to 14 days was characterized. During 3'-MeDAB hepatocarcinogenesis, the two moieties of the carcinogen-protein complex have considerably different turnover rates. With continued ingestion of the azocarcinogen, the bound azo dye in the complex has a half-life of 2.5 days, but the protein moiety has a half-life of 8.7 days. In addition, the interaction of 3'-MeDAB with the principal target protein in vivo extends the half-life of the protein itself from 3.3 days in normal liver to 8.7 days. The slowing of the turnover of the protein by the carcinogen appears to be readily reversible, since the half-life of the protein returns to that of the target protein in normal liver soon after the end of the azo dye feeding. The considerable difference in turnover rates of the two moieties of the complex and the reversible effects of the carcinogen in slowing the turnover of the protein moiety suggest that the two moieties of the native azoprotein are noncovalently linked and that they have different biological activities. The native complex contains azo dye in an activated state that is capable of yielding a reactive electrophile; after protein denaturation, the bound azo dye was previously found to have properties indicative of covalent linkage to the protein. The retardation in the biological turnover rate of the protein moiety, apparently resulting from interaction with azocarcinogen, agrees with the known ligand-induced stabilization in vitro and reduced rate of proteolytic degradation in vivo of other proteins that result from conformational change to a more compact configuration. The evidence is consistent with the hypothesis that the principal liver carcinogen-protein complex contains hydrophobically bound activated azocarcinogen, whose specificity of reaction with critical macromolecule(s) in nuclei may be directed by the conformationally altered protein of an activated carcinogen-protein complex, ie, a specific receptor protein containing activated azocarcinogen. (24 refs.)

**77-2475 Inhibition of Chemical Carcinogenesis: Increased Activity of Soluble RNA Polymerase in the Liver of Rats Protected Against 3'MeDAB Hepatocarcinogenesis by Dietary Chloramphenicol.** (Eng.) Phillips, W. A. (Dept. Pathology, Univ. Melbourne, Parkville, Victoria 3052, Australia) Blunck, J. *Eur J Cancer* 13(7): 729-747; 1977.

Soluble RNA polymerases were isolated from the liver nuclei of male Sprague-Dawley rats pair-fed diets containing the hepatocarcinogen 3' methyl-4-dimethylaminoazobenzene (3'MeDAB, 0.06%, alone or in combination with chloramphenicol (CAP, 2%), an inhibitor of azo dye carcinogenesis. DEAE Sephadex A-25 chromatography of crude nuclear extracts (fraction IV protein) revealed two major peaks of enzyme activity that were equated with RNA polymerases I and II on the basis of order of elution and sensitivity to amanitin. The specific activities of both enzymes, in particular RNA polymerase I, were significantly increased after 4 days on diets containing CAP or 3'MeDAB + CAP. Rats protected from 3'MeDAB carcinogenesis by CAP showed the greatest increase, which exceeded that in the group fed dye alone by 292% for polymerase I and 116% for polymerase II. Increases in both RNA polymerase activities were probably not a consequence of altered affinity for the substrate. RNA polymerase activity was not altered by feeding the 3'MeDAB diet for 4 days, but after 10 days there was a 340% increase in the specific activity of enzyme I and a 14% increase in that of enzyme II relative to control levels. The total nuclear protein content increased significantly in rats fed 3'MeDAB or 3'MeDAB + CAP for 4 days. Crude polymerase extracts contained 4%-6% of the total nuclear protein and 83%-87% of the RNA polymerase activity present in the isolated nuclei. There was a significant increase in the amount of nuclear sap protein extracted after feeding 3'MeDAB for 4 days. Differences in nuclear RNA synthesis between experimental groups were insensitive to presacrifice starvation and altered feeding and lighting schedules. They were also not due to differential losses of RNA polymerases during the nuclear isolation procedure or to preferential extraction of these enzymes from isolated nuclei in the various groups. It is concluded that the differences in liver nuclear RNA synthesis between 3'MeDAB-fed rats and rats protected against 3'MeDAB-induced hepatocarcinogenesis by concurrent CAP administration are at least partly due to differences in the activity and/or amount of soluble-RNA polymerases. (84 refs.)

**77-2476 Enhancement of Azo-Dye Hepatocarcinogenesis with Dietary Phenobarbital in Rats.** (Eng.) Kitagawa, T. (Dept. Pathology, Cancer Inst., Kami-Ikebukuro 1-37-1, Toshima-ku, Tokyo 170, Japan) Sugano, H. *Gann* 68(2): 255-256; 1977.

The enhancing effect of phenobarbital (PB) in azo-dye carcinogenesis was studied by examining the number and size

of the resulting carcinomas and the number of ATPase-deficient islands during carcinogenesis. Male Donryu rats were fed a diet containing 0.06% 3'-methyl-4-(dimethylamino)azobenzene (3'-Me-DAB) for 3 wk starting at 21 days of age. After 2 wk, one group of rats was maintained on the same diet and a second group was given a diet containing 0.05% PB. Rats were sacrificed at 6, 8, 12, and 24 wk. The number of carcinomas per rat was 14 times greater in the group fed PB than in the group maintained on the 3'-Me-DAB diet alone. The size of the carcinomas was also larger in PB-fed rats. All tumors were well-differentiated carcinomas except for one poorly differentiated type in the PB group. The number and size of ATPase-deficient islands were also significantly larger in the livers from the PB rats. In both groups these parameters increased rapidly during 6-12 wk. In the PB group, there was a further increase during 12-24 wk, but in the other group, the number and size of these enzyme-deficient islands remained relatively constant. These findings indicate that PB actually increases the potential for cancer production rather than merely accelerates the cancerous process in lesions which are already preneoplastic. case. (7 refs.)

**77-2477 The Histochemical Demonstration of Polycytidylic Acid and Polyuridylic Acid Hydrolases in Rat Liver During Azo Dye Carcinogenesis.** (Eng.) Daoust, R. (Institut du Cancer de Montreal, Centre Hospitalier Notre-Dame, Montreal, Canada) *J Histochem Cytochem* 25(6): 458-465; 1977.

Films of polycytidylic acid (poly-C) and polyuridylic acid (poly-U) were exposed to liver sections from male Wistar rats fed a control diet or a diet containing 4-dimethylaminoazobenzene (DAB). The results were compared with those obtained with films of RNA. Livers from rats fed the basal control diet showed a distribution of poly-C and poly-U hydrolase activities comparable to that observed in normal tissue but they exhibited a significant increase in centrilobular ribonuclease activity. In livers of DAB-fed rats, the centrilobular necrotic tissue was negative to poly-C and strongly positive to poly-U and RNA. The regenerating parenchyma gave positive reactions with the different substrates, but areas of the hyperplastic tissue became deficient in all three hydrolase activities after 8 wk of azo dye feeding. The hyperbasophilic foci that developed a few wk later were inactive against poly-C, poly-U, and RNA; the hepatomas were also negative except for the necrotic and the stromal tissues, which gave positive reactions with poly-U and RNA. These results confirm that different nucleases can be demonstrated histochemically by the use of films of polyribonucleotides and suggest that tumors derive from nuclease-deficient areas of preneoplastic tissue. (18 refs.)

**77-2478 The Effect of Propranolol on Induction of Rat Liver Tumors by a Chemical Carcinogen.** (Eng.)



Boyd, H. (Dept. Pharmacology, Univ. Miami Medical Sch., Miami, FL 33152) Martin, T. J. *Mol Pharmacol* 13(3): 576-578; 1977.

To investigate what role the increased beta adrenergic responsiveness of adenylate cyclase might play in neoplastic transformation, the effect of the beta adrenergic blocker propranolol on tumor incidence in rat liver was investigated during treatment with the carcinogen 3'-methyl-4-dimethylaminoazobenzene. High doses (0.02-0.5%) of propranolol in the drinking water approximately doubled tumor incidence. These results showed that increased beta adrenergic responsiveness does not promote carcinogenesis. Whether propranolol facilitated neoplastic transformation in this system by specific beta blockade or by nonspecific actions remains to be elucidated. (10 refs.)

**77-2479 Study of Rat Liver Nuclear RNA Population After a Short-term Carcinogen Treatment.** (Rus.) Barskaya, T. V. (Lab. Genetics Tumor Cells, Inst. Cytology Academy Sciences, Leningrad, USSR) Zakharova, N. V.; Olenov, Iu. M. *Tsitologiya* 19(2): 235-238; 1977.

To determine the rat liver nuclear RNA population after one injection of 4-dimethylaminoazobenzene, RNA-DNA hybridization in a solution with DNA excess was employed. After 24 hr the treated livers were compared with livers from normal untreated rats, and an altered transcription was demonstrated. The hybridization capacity of the nuclear RNA isolated from carcinogen-treated livers was lower than that of the control RNA. These changes may be due to changes in the rate of transcription in DNA sites. (11 refs.)

**77-2480 Studies on the Inhibition of Transcription by the Hepatocarcinogen N-Hydroxy -2- acetylaminofluorene.** (Eng.) Austin, G. E.; Roop, B.; Schwartz, E.; Moyer, G. H. In: *Molecular Mechanisms in the Control of Gene Expression. ICN-UCLA Symposia on Molecular and Cellular Biology*. Nierlich, D. P.; Rutter, W. J.; Fox, C. F., eds. (New York: Academic Press, Inc.): Vol. 5, pp. 273-278; 1976.

The mechanisms of inhibition of hepatic RNA synthesis by the carcinogen N-hydroxy-2-acetylaminofluorene (N-OH AAF) were examined in male Sprague-Dawley rats given a single dose of N-OH AAF (30 mg/kg) ip in propylene glycol. RNA polymerase II was inhibited by 70% in the N-OH AAF-treated animals and polymerase I by 40%-50% according to the results of diethylaminoethyl Sephadex chromatography. There was a rapid inhibition of protein synthesis in N-OH AAF-treated animals, suggesting that such a mechanism contributed to the extensive inhibition of ribosomal RNA synthesis by the carcinogen. Chromatin preparations from control rats and animals sacrificed 2 hr after administra-

tion of N-OH AAF possessed identical template activity for transcription by rat liver RNA polymerase. Studies of the effects of in vitro arylamidation by the reactive ester N-acetoxy AAF indicated that binding of carcinogen to enzyme protein in vivo was not sufficient to account for the observed degree of enzyme inhibition. It is possible that secondary factors in vivo may sensitize the enzyme to inactivation following minor damage by N-OH AAF. (8 refs.)

**77-2481 Procarcinogen Activation and Hormonal Control of Cell Proliferation in Differentiated Primary Adult Rat Liver Cell Cultures.** (Eng.) Leffert, H. L. (Cell Biology Lab., Salk Inst., Post Office Box 1809, San Diego, CA 92112) Moran, T.; Boorstein, R.; Koch, K. S. *Nature* 267(5606): 58-61; 1977.

Procarcinogen activation and hormonal control of cell proliferation in differentiated primary adult rat liver cells were studied with a novel primary monolayer culture system. Cells were isolated for culturing from 5-7 g of normal liver using arginine-free medium to select for arginine-synthesizing hepatocytes. Studies of growth kinetics showed that serum alone failed to sustain significant proliferation rates, but if hormones and inosine were added, proliferation was stimulated. Titration studies suggested synergistic hormone interactions. Triiodothyronine and glucagon did not stimulate growth. The stimulated cells maintained differentiated hepatic functions such as albumin and arginine biosynthesis for at least 8-10 days after plating. They also metabolized the procarcinogen N-2-acetylaminofluorene (AAF) into a proximate form, N-hydroxy-AAF. This process appears to be liver-cell-specific and is performed by viable cells, not by extracellular chemical and/or enzymatic mechanisms. The critical interactions required of hormones, nutrients, and carcinogens to alter proliferative control are unknown. (20 refs.)

**77-2482 Arylhydroxamic Acid Acyltransferase (AT)-Catalyzed Induction of Mutations in *Salmonella Typhimurium* (Meeting Abstract).** (Eng.) Weeks, C. E. (NCTR, FDA, Jefferson, AR 72079) Allaben, W. T.; Louie, S. C. *Proc Am Assoc Cancer Res* 18: 122; 1977. (no refs.)

**77-2483 Preneoplastic Antigen as a Marker for Endoplasmic Reticulum of Putative Premalignant Hepatocytes During Liver Carcinogenesis.** (Eng.) Lin, J. C. (Dept. Pathology, Univ. Toronto, Medical Sciences Building, Toronto, Ontario, Canada M5S 1A8) Hiasa, Y.; Farber, E. *Cancer Res* 37(7): 1972-1981; 1977.

The subcellular localization of a preneoplastic antigen in hyperplastic liver nodules and primary hepatocellular carcinomas induced in Fischer rats by 0.02% 2-

2-acetylaminofluorene (2-AAF) was studied further. Rabbit antisera against four subcellular fractions, cytosol, smooth and rough endoplasmic reticulum (SER, RER), and free lysosomes, were used to assay for the antigen by immunodiffusion. The preneoplastic antigen, antigen 1, which appears as a sharp precipitin line, is located predominantly, if not exclusively, in the SER fraction of hyperplastic nodules and hepatomas. It appears after about 3 wk of 2-AAF feeding and persists throughout the carcinogenic process in nodules and hepatomas. A second antigen, antigen 2, appears in the SER of nodules after 13 wk of 2-AAF feeding but gradually disappears on discontinuation of exposure to the carcinogen after 10 wk. This antigen, appearing as a more diffuse precipitin line, becomes demonstrable in RER as well, but only after the ribosomes are stripped off. The presence and distribution of preneoplastic antigen during carcinogenesis, as revealed by immunodiffusion, were similar when a more sensitive assay, microcomplement fixation, was used. The preneoplastic antigen appears to be a potentially useful marker for alterations in the SER that may be related to the development of liver cancer. (29 refs.)

77-2484 Rat  $\alpha$ -Macrofetoprotein During Hepatocarcinogenesis (Meeting Abstract). (Eng.) Hudig, J. D. (Univ. California, San Diego, La Jolla, CA 92093) Sell, S.; Becker, F. F. *Proc Am Assoc Cancer Res* 18: 128; 1977. (no refs.)

77-2485 Quantitative Histochemical and Autoradiographic Studies of 2-Acetylaminofluorene (2-AAF) Hepatocarcinogenesis (Meeting Abstract). (Eng.) Pugh, T. D. (Univ. Wisconsin, Madison, WI 53706) Goldfarb, S. *Proc Am Assoc Cancer Res* 18: 130; 1977. (no refs.)

77-2486 Analysis of 2-Acetylaminofluorene Metabolites in Biological Systems Using Liquid Chromatography (Meeting Abstract). (Eng.) Stanley, J. W. (NCTR, Jefferson, AR 72079) *Proc Am Assoc Cancer Res* 18:156; 1977. (no refs.)

77-2487 In Vitro Transformation of Diploid Human Cells with Chemical Carcinogens (Meeting Abstract). (Eng.) Milo, G. E. (Dept. Physiology, Chemistry, and Veterinary Pathobiology, Ohio State Univ., Columbus, OH 43210) DiPaolo, J. A. *In Vitro* 13(3): 193; 1977. (no refs.)

77-2488 Changes in Ploidy of Rat Liver Nuclei During Carcinogenesis with 2-Acetylaminofluorene (Meeting Abstract). (Eng.) Roszell, J. A. (Veterans Admin. Hosp. and Univ. Tennessee Center for the Health Sciences, Memphis, TN 38104) Fredi, J. L.; Irving, C. C. *Proc Am Assoc Cancer Res* 18: 167; 1977. (no refs.)

77-2489 N-Acetoxy-2-acetylaminofluorene Inhibits Repair of Damaged DNA in Chinese Hamster Cells (Meeting Abstract). (Eng.) Ahmed, F. E. (Biology Dept., Brookhaven Natl. Lab., Upton, NY 11973) Setlow, R. B. *Biophys J* 17(2): 245; 1977. (no refs.)

77-2490 N-Acetoxy-Acetylaminofluorene Enhances the Rate of Postreplication Repair in Xeroderma Pigmentosum Variant Cells. (Eng.) D'Ambrosio, S. M. (Biology Dept., Brookhaven Natl. Lab., Upton, NY 11973) Setlow, R. B. *Fed Proc* 36(3): 848; 1977. (no refs.)

77-2491 DNA Repair in Primary Cultures of Rat Hepatocytes (Meeting Abstract). (Eng.) Yager, J. D. (Dept. Pathology, Dartmouth Medical Sch., Hanover, NH 03755) Miller, J. A. *Proc Am Assoc Cancer Res* 18: 159; 1977. (no refs.)

77-2492 Rat Liver Homogenate-mediated Toxicity and Induction of 6-Thioguanine-resistance in V79 Chinese Hamster Cells by Chemical Carcinogens (Meeting Abstract). (Eng.) Krahn, D. F. (Univ. Wisconsin, Madison, WI 53706) *Diss Abstr Int [B]* 37(8): 3726; 1977. (no refs.)

77-2493 Early Functional and Histochemical Properties of Carcinogen-Altered Hepatocytes (Meeting Abstract). (Eng.) Solt, D. (Dept. Pathology, Univ. Toronto, Toronto, Ontario, Canada, M5S 1A8) Ogawa, K.; Farber, E. *Lab Invest* 36(3): 350-351; 1977. (no refs.)

77-2494 Synergistic Effect of Urinary Bladder Carcinogenesis in Rats Treated with N-Butyl-N-(4-hydroxybutyl)nitrosamine, N-[4-(5-Nitro-2-furyl)-2-thiazolyl]formamide, N-2-Fluorenylacetamide, and 3,3'-Dichlorobenzidine. (Eng.) Tsuda, H. (First Dept. Pathology, Nagoya City Univ. Medical Sch., 1 Kawasumi, Mizuho-cho,



Mizuho-ku, Nagoya 467, Japan) Miyata, Y.; Murasaki, G.; Kinoshita, H.; Fukushima, S.; Ito, N. *Gann* 68(2): 183-192; 1977.

The possible synergistic effects of N-butyl-N-(4-hydroxybutyl)nitrosamine (BBN), N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT), N-2-fluorenylacetamide (2-FAA), and 3,3'-dichlorobenzidine (3,3'-DCB) were investigated in the urinary bladder of male Wistar rats. The chemicals were administered singly or two or three together at non-carcinogenic doses in the diet for 40 wk, and resulting changes in the urinary bladder were examined. The incidence of focal hyperplasia of the bladder epithelium was significantly higher in groups treated with BBN + FANFT, BBN + 3,3'-DCB, BBN + 2-FAA, and BBN + 3,3'-DCB + 2-FAA than in those treated with a single chemical. It was also higher in the group treated with 2-FAA + FANFT than in the group treated with 2-FAA alone. The incidence of papilloma was higher in the group treated with BBN + 2-FAA than in rats treated with 3BN, FANFT, or 2-FAA alone. The incidence of transitional cell carcinoma was higher in the group treated with BBN + FANFT than in the group treated with BBN only. Liver lesions developed in rats treated with 2-FAA alone or in combination with BBN or FANFT, but they were rare after treatment with 3,3'-DCB + 2-FAA. These findings illustrate the synergistic effects of BBN with FANFT, 2-FAA, and/or 3,3'-DCB on bladder tumorigenesis. The effects of BBN and FANFT were most markedly synergistic. (43 refs.)

**77-2495 Free Radicals Produced in a Nitrosofluorene-unsaturated Lipid Reaction.** (Eng.) Floyd, R. A. (Biomembrane Res. Lab., Oklahoma Medical Res. Foundation, 825 Northeast 13th St., Oklahoma City, OK 73104) *Experientia* 33(2): 197-198; 1977.

2-Nitrosofluorene, an activated form of 2-acetylaminofluorene, was found to react with lipid molecules containing carbon-carbon double bonds yielding free radicals. The amount of free radicals tended to follow the number of double bonds in the lipid. (12 refs.)

**77-2496 Microsomal N-Hydroxylase Modification as a First Step in Carcinogenesis by Arylamines** (Meeting Abstract). (Eng.) Roberfroid, M. (Laboratoire de Biotoxicologie, Ecole de Pharmacie, U.C.L.-73.69, Avenue Emmanuel Mounier, 1200 Bruxelles, Belgium) Razzouk, C.; Mercier, M. *Arch Int Physiol Biochim* 85(2): 429-431; 1977. (5 refs.)

**77-2497 Studies on Induction of Rat Liver Microsomal Membrane Polypeptides by Hepatocarcinogens**

(Meeting Abstract). (Eng.) Cameron, R. (Univ. Toronto, Toronto, Ontario M5S 1A8, Canada) Sharma, R. N.; Murray, R. K. *Proc Am Assoc Cancer Res* 18: 122; 1977. (no refs.)

**77-2498 Obligatory Free Radical Intermediate in the Oxidative Activation of the Carcinogen N-Hydroxy-2-acetylaminofluorene.** (Eng.) Floyd, R. A. (Biomembrane Res. Lab., Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104) Soong, L. M. *Biochim Biophys Acta* 498(1): 244-249; 1977.

The products of the hematin-cumene hydroperoxide oxidative activation of the carcinogen N-hydroxy-2-acetylaminofluorene (OH-AAF) were characterized by thin-layer chromatography and by kinetic experiments involving optical and electron spin resonance techniques. The nitroxyl free-radical form of OH-AAF is an obligatory intermediate in the oxidative activation of this carcinogen into 2-nitrosofluorene and N-acetoxy-2-acetylaminofluorene. Both the rate of N-OH-acetylaminofluorene oxidation and the amount of its nitroxyl free radical were a function of reaction time. Rate equations were derived for a model in which the nitroxyl free-radical form of OH-AAF was an obligatory intermediate in the reaction. With this theory it was possible to compute one experimental variable, the rate of OH-AAF oxidation, utilizing the other experimental variable, the amount of nitroxyl free radical present at any time during the reaction. The theory also predicts a linear relationship between the rate of OH-AAF oxidation and the square of the free-radical content; this was confirmed experimentally. The dismutation rate constant of the nitroxyl free radical of OH-AAF was  $2.7 \times 10^5/\text{M}/\text{sec}$ . (14 refs.)

**77-2499 Quantitative Studies of Transformation by Chemical Carcinogens and Ultraviolet Radiation Using a Subclone of BHK<sub>21</sub>, Clone 13 Syrian Hamster Cells.** (Eng.) Ishii, Y. (Brookhaven Natl. Lab., Upton, NY 11973) Elliott, J. A.; Mishra, N. K.; Lieberman, M. W. *Cancer Res* 37(7): 2023-2029; 1977.

Transformation (growth in soft agar) and survival of a subclone of BHK<sub>21</sub>, C13 Syrian hamster cells were evaluated after treatment with N-acetoxy-2-acetylaminofluorene (N-AcO-AAF), N-methyl-N'-nitro-N-nitrosoguanidine (MNNG), 4-nitroquinoline 1-oxide (4NQO), and UV radiation. Because cells of this subclone are capable of dividing several times in soft agar before dying, transformation is expressible in these cells without the intermediate step of plating in liquid media with a solid plastic or glass substrate. Thus, a quantitative assessment of transformation can be obtained by treating these cells and then trypsinizing them and plating them directly in soft agar. The spontaneous transformation frequency of this subclone is  $5$  to  $10 \times 10^{-6}$ . As a function of molar dose, 4NQO is the most effective at inducing transformation,

and N-AcO-AAF and MNNG are about equally effective. The precarcinogen acetylaminofluorene, at concentrations up to 20  $\mu$ M, did not increase the frequency of transformants. If transformation is scored on the basis of number of cells inoculated and under conditions of high relative survival, the number of transformants increases with concentration of chemical or influence of UV radiation. These data indicate that the agents induce transformation rather than select for existing transformants. If transformation is scored as a function of survival, MNNG and 4NQO are the most effective transforming agents, N-AcO-AAF is intermediate, and UV radiation gives the lowest transformation frequency. Isolation of transformed colonies (both induced and spontaneous) and sc injection into Syrian hamsters resulted in tumors from about one-half of the isolated clones. Untreated cells produced no tumors under these conditions. Both untransformed and transformed cells were free of a variety of viruses. (29 refs.)

77-2500  $\gamma$ -Glutamyl Transferase in Putative Premalignant Hepatocyte Populations During Hepatocarcinogenesis Induced by Diethylnitrosamine: A Biochemical Study (Meeting Abstract). (Eng.) Cameron, R. (Univ. Toronto, Dept. Pathology, Toronto, Ontario, Canada, M5S 1A8) Kellen, J.; Malkin, A.; Kolin, A. *Lab Invest* 36(3): 332; 1977. (no refs.)

77-2501  $\gamma$ -Glutamyltranspeptidase (GGT) as a Very Early Marker of Putative Preneoplastic Cells in Liver Carcinogenesis (Meeting Abstract). (Eng.) Ogawa, K. (Dept. Pathology, Univ. Toronto, Ontario, Canada) *Proc Am Assoc Cancer Res* 18: 158; 1977. (no refs.)

77-2502  $\gamma$ -Glutamyl Transpeptidase: A Positive Marker for Cultured Rat Hepatocytes Derived from Putative Premalignant and Malignant Lesions (Meeting Abstract). (Eng.) Laishes, B. A. (Univ. Toronto, Toronto, Ontario, Canada, M5S 1A8) *Proc Am Assoc Cancer Res* 18: 140; 1977. (1 ref.)

77-2503 Aflatoxins in Egyptian Foodstuffs. (Eng.) Girgis, A. N. (Natl. Organization for Drug Control and Res., Cairo, Egypt) El-Sherif, S.; Rofael, N.; Nesheim, A. *J Assoc Off Anal Chem* 60(3): 746-747; 1977.

Thin-layer chromatographic analyses of fresh and stored Egyptian foodstuffs showed that aflatoxins B<sub>1</sub>, B<sub>2</sub>, G<sub>1</sub> and G<sub>2</sub> were present in 14/42 samples at low levels (3-12 ppb). The

highest levels were in cottonseed cake. Analyses by the minicolumn procedure gave positive results for only 5 samples, negative results for 31 samples, and indefinite results for 6 samples. (4 refs.)

77-2504 Aflatoxin in Tunisian Aleppo Pine Nuts. (Eng.) Boutrif, E. (Institut National de Nutrition et de Technologie Alimentaire, Tunis, Tunisia) Jemmali, M.; Pohland, A. E.; Campbell, A. D. *J Assoc Off Anal Chem* 60(3): 747-748; 1977.

Aflatoxin levels in contaminated Tunisian Aleppo pine nuts (25/50 samples) varied from 25 to 2,080 ppb for aflatoxin B<sub>1</sub> and from 56 to 4,570 ppb for aflatoxin G<sub>1</sub>. Total aflatoxin reached 7,550 ppb in some samples. A traditional pudding prepared from the contaminated nuts contained 83% of the aflatoxin present in the nuts. (4 refs.)

77-2505 Toxicological Evaluation of the Mycotoxin, Sterigmatocystin, An Environmental Carcinogen (Meeting Abstract). (Eng.) Moore, M. R. (Univ. Texas at Austin, Austin, TX 78712) *Diss Abstr Int [B]* 37(8): 3904-3905; 1977. (no refs.)

77-2506 Effects of Dietary Feeding of Aflatoxin B<sub>1</sub> on Ribosomal RNA Metabolism in Rat Liver. (Eng.) Smith, S. J. (Dept. Pharmacology, Pennsylvania State Univ., Milton S. Hershey Medical Center, Hershey, PA 17033) Deen, K. C.; Calhoun, W. J.; Beittenmiller, H. F. *Cancer Res* 37(7): 2226-2231; 1977.

After 1 wk of feeding aflatoxin B<sub>1</sub> (AFB<sub>1</sub>, 1 or 10 ppm in the diet) to male Fischer rats, sucrose gradient analysis revealed decreases in the in vivo incorporation of <sup>3</sup>H-orotic acid into 45S hepatic nuclear RNA (nRNA) and a decreased concentration (A<sub>260</sub>) of 28S nRNA when compared with controls fed an identical diet minus AFB<sub>1</sub>. Sucrose gradient analysis of hepatic ribosomal (rRNA) showed decreases of a similar magnitude in labeling in vivo and in A<sub>260</sub> of 28S rRNA. Labeling and A<sub>260</sub> of the 18S rRNA were unchanged. Assay of RNA polymerase I activity in isolated hepatic nuclei demonstrated that this enzyme activity was not diminished in rats fed the AFB<sub>1</sub> diet from that of controls. Feeding of AFB<sub>1</sub> for 1-6 wk resulted in progressive decreases in A<sub>260</sub> of 28S nRNA and in both label and A<sub>260</sub> in microsomal 28S rRNA. These effects are time- and dose-related, since 1 wk of a diet containing 10 ppm produced changes in nuclear and ribosomal 28S RNA similar to those observed after 4-6 wk of a diet containing 1 ppm. Sixteen hours after a single injection of AFB<sub>1</sub> (1 mg/kg, ip), the same defects in RNA metabolism occurred as those described for 1 ppm for 4-6 wk and 10 ppm for 1



wk. In contrast to the chronic feeding studies, after an acute injection these effects eventually disappeared. These data suggest that early progressive lesions in the maturation of hepatic 28S rRNA are produced during chronic feeding of a diet containing AFB<sub>1</sub>. Such defects in the processing of ribosomal precursor RNA may be a characteristic feature of chemical hepatocarcinogenesis. (52 refs.)

**77-2507 Structural Identification of the Major DNA Adduct Formed by Aflatoxin B<sub>1</sub> In Vitro. (Eng.)**

Essigmann, J. M. (Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA 02139) Croy, R. G.; Nadzan, A. M.; Busby, W. F.; Reinhold, V. N.; Buchi, G.; Wogan, G. N. *Proc Natl Acad Sci USA* 74(5): 1870-1874; 1977.

Aflatoxin B<sub>1</sub> (AFB<sub>1</sub>) was activated metabolically by rat liver microsomes and bound to calf thymus DNA in vitro in order to isolate and analyze the structure of the bound form of the carcinogen. The bound intermediate was removed from DNA with formic acid, and 90% of the carcinogen was shown to be a single component by reverse-phase high-pressure liquid chromatography. Larger quantities of the adduct were isolated by high-pressure liquid chromatography, and they were subjected to chemical, nuclear magnetic resonance, UV, and mass spectral analysis. The results showed that the DNA-bound product was 2,3-dihydro-2-(N<sup>7</sup>-guanylyl)-3-hydroxyaflatoxin B<sub>1</sub>. Identification was also confirmed by correlating the UV spectra with those of a model compound. The structural data support indirect evidence that aflatoxin B<sub>1</sub> 2,3-oxide is quantitatively important as an intermediate in the binding of AFB<sub>1</sub> to nucleic acids. (36 refs.)

**77-2508 Human Metabolism of Aflatoxin B<sub>1</sub>: Comparisons Among Individuals for Patterns of Hepatic Detoxification (Meeting Abstract). (Eng.)** Yourtee, D. M. (Dept. Biochemistry, Cancer Res. Center, Columbia, MO 65201) Phillips, D. L.; Flood, M. H. *Biophys J* 17(2): 258; 1977. (no refs.)

**77-2509 Liver Homogenate-mediated Mutagenesis in Chinese Hamster V79 Cells by Polycyclic Aromatic Hydrocarbons and Aflatoxins. (Eng.)** Krahn, D. F. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI 53706) Heidelberg, C. *Mutat Res* 46(1): 27-44; 1977.

A mammalian cell culture mutagenesis assay using Chinese hamster V79 cells, which are sensitive to the cytotoxic and mutagenic effects of several chemical carcinogens that require metabolic activation, is described. The induced frequen-

cy of 6-thioguanine-resistant colonies was used to measure mutagenic activity. The 9000-g supernatant fraction of rat liver plus cofactors provided the metabolic activation. Eventually, the assay could be utilized to prescreen environmental chemicals. The following chemical carcinogens were examined: aflatoxin B<sub>1</sub>, aflatoxin B<sub>2</sub>, benzo(a)pyrene, 3-methylcholanthrene, 7,12-dimethylbenz(a)anthracene, dibenz(a,h)anthracene, dibenz(a,c)anthracene, and benz(a)anthracene. Except for dibenz(a,h)-anthracene and dibenz(a,c)-anthracene, the mutagenic activity generally paralleled the carcinogenic activity. (64 refs.)

**77-2510 Properties of Carcinogen-Altered Hepatocytes in Cell Culture (Meeting Abstract). (Eng.)**

Laishes, B. (Dept. Pathology, Univ. Toronto, Toronto, Ontario, Canada, M5S 1A8) Farber, E. *Lab Invest* 36(3): 357-358; 1977. (no refs.)

**77-2511 Mammary Tumor and Leukemia in Male Sprague-Dawley Rats Evoked by a Series of Intragastric Administration of 7,12-Dimethylbenz(a)anthracene. (Eng.)** Yoshida, H. (Dept. Pathology, Ehime Univ. Sch. Medicine, Shizukawa, Shigenobu-cho, Onsen-gun, Ehime-ken 791-02, Japan) Fukunishi, R. *Gann* 68(2): 237-239; 1977.

Tumor induction by a series of intragastric doses of 7,12-dimethylbenz(a)anthracene (DMBA) was studied in male Sprague-Dawley rats. DMBA was given in eight 10-mg doses, the first at 28 days of age followed by seven at 14-day intervals. Before they had received the eight doses, 147/311 rats died of weakness with hypo- or aplastic bone marrow. The incidence of cancer was as follows: mammary tumors, 52.4%; leukemia 18.9%, and ear duct cancer 7.3%. The mammary tumors were mostly papillotubular adenocarcinomas. Of the leukemias, 16/31 were of the erythroblastic stem cell type and 15/31 were myelogenous. Liver invasion was extensive in both types of leukemia. The majority of ear duct tumors had superficial ulceration of the skin, and they were classified histologically as keratinizing epidermoid carcinomas. These results are different from those of a similar study using Long-Evans rats, particularly in the incidence of tumors of the mammary glands and hematopoietic organs. The results indicate that the incidence and type of induced tumors are different in different strains of rats under the same treatment. (17 refs.)

**77-2512 Modification of Superoxide Dismutase in Rat Mammary Carcinoma. (Eng.)** Petkau, A. (Medical Biophysics Branch, Whiteshell Nuclear Res. Establishment, Atomic Energy Canada Limited, Pinawa, Manitoba, Canada ROE 1LO) Monasterski, L. G.; Kelly, K.; Friesen,

H. G. *Res Commun Chem Pathol Pharmacol* 17(1): 125-132; 1977.

Protein and superoxide dismutase levels were assayed in the normal mammary tissue of Sprague-Dawley rats and in mammary carcinomas induced by 7,12-dimethylbenz(a)anthracene (DMBA, 5 mg/g of emulsion). The tissue protein was distributed homogeneously and increased significantly in the mammary carcinomas. At the center and margin of the carcinoma, the mean concentration of superoxide dismutase was approx 54 and 117  $\mu\text{g/g}$ , respectively, but in the tumor as a whole it was 104  $\mu\text{g/g}$ . The latter value was not significantly different from the concentration in the mammary tissue of lactating rats (approx 113  $\mu\text{g/g}$ ). Exposure of the tumor-bearing rats to hyperoxia did not increase the tumor protein, but it increased the enzyme concentration at the center and margin of the carcinoma to 162 and 86  $\mu\text{g/g}$ , respectively. (16 refs.)

7-2513 **Effect of Hormones on the Survival of Mammary Tumours In Vitro (Meeting Abstract).** (Eng.) Kesava Rao, K. V. (Biology Div., Cancer Res. Inst., Tata Memorial Centre, Parel, Bombay 400 012 India) Pai, S. R.; Bhat, A. V.; Bapat, C. V. *In Vitro* 13(3): 205; 1977. (no refs.)

7-2514 **Nodule Like Alveolar Lesions (NLAL) Induced by DMBA and Hormonal Influence on Its Binding to Mammary DNA in Organ Culture (Meeting Abstract).** (Eng.) Telang, N. T. (Tumor Biology Lab., Sch. Life Sciences, Univ. Nebraska, Lincoln, NB 68588) Kundu, A. B.; Rump, L. R.; Banerjee, M. R. *Proc Am Assoc Cancer Res* 18: 118; 1977. (no refs.)

7-2515 **Effects of Adrenergic Agonists and Antagonists on Growth of DMBA-Induced Mammary Tumors and Serum Prolactin Levels (Meeting Abstract).** (Eng.) Rodson, C. A. (Dept. Physiology, Endocrine Res. Lab., Michigan State Univ., East Lansing, MI 48824) Mioduszewski, J.; Meites, J. *Proc Am Assoc Cancer Res* 18: 159; 1977. (no refs.)

7-2516 **In Vitro Growth Characteristics of Epithelial Cell Lines Derived from Tracheal Transplants Exposed In Vivo to 7,12-Dimethylbenz(a)anthracene (DMBA) (Meeting Abstract).** (Eng.) Marchok, A. C. (Cancer and Toxicology Program, Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Nettesheim, P. *In Vitro* 13(3): 193; 1977. (no refs.)

77-2517 **Photodynamic Enhanced Killing of V79 Chinese Hamster Cells Exposed to "Sunlight" in the Presence of 7,12-Dimethylbenz(a)anthracene (Meeting Abstract).** (Eng.) Elkind, M. M. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439) Utsumi, H. *Proc Am Assoc Cancer Res* 18: 162; 1977. (no refs.)

77-2518 **A Study of the Primary Effect of Exposure to Carcinogens (Activity and Isoenzyme Composition of Enzymes).** (Rus.) Khodosova, I. A. (Lab. Tumor Cell Genetics, Inst. Cytology, Acad. Sciences USSR, Leningrad, USSR) Bozhkov, V. M.; Avertsev, S. A.; Olenov, Iu. M. *Vestn Akad Med Nauk SSSR* (3): 17-19; 1977.

Exposure to polycyclic hydrocarbons, azocompounds and nitrosamines produced similar changes in the enzymatic activity of a given target tissue. The activity of the carcinogen did not correlate with the extent of the induced changes. (4 refs.)

77-2519 **Surface Changes in Chinese Hamster Cells During Their Progression in Culture Following Carcinogen Treatment (Meeting Abstract).** (Eng.) Harrison, C. J. (Paterson Lab., Christie Hosp. and Holt Radium Inst., Manchester M20 9BX, England) Allen, T. D. *Br J Cancer* 35(2): 255; 1977. (no refs.)

77-2520 **Changes in Chinese Hamster Cells During Their Progression in Culture Following Carcinogen Treatment (Meeting Abstract).** (Eng.) Connell, J. R. (Paterson Lab., Christie Hosp. and Holt Radium Inst., Manchester M20 9BX, England) Ockey, C. H. *Br J Cancer* 34(2): 249; 1977. (no refs.)

77-2521 **In Vivo Programmed Cell Death by Differentiation of Malignant Epithelial Cells (Meeting Abstract).** (Eng.) Singh, B. (Medical Coll. Georgia, Augusta, CA 30902) McKinney, R.; Brewer, P. *Proc Am Assoc Cancer Res* 18: 165; 1977. (1 ref.)

77-2522 **Exceptional Mutagenicity of Bay Region Epoxides of Benzo(a)anthracene 3,4-Dihydrodiol (Meeting Abstract).** (Eng.) Wood, A. W. (Hoffmann-La Roche Inc., Nutley, NJ 07110) Chang, R. L.; Levin, W.; Lehr, R. E.; Ridder, M. S.; Karle, J. M.; Jerina, R. E.; Conney, A. H. *Proc Am Assoc Cancer Res* 18: 119; 1977. (1 ref.)



77-2523 Latent Period Reduction of 1,2-Dimethylhydrazine Carcinogenesis in Mice by a Variant of *Citrobacter freundii* (Meeting Abstract). (Eng.) Barthold, S. W. (Yale Univ. Sch. Medicine, New Haven, CT 06510) *Lab Invest* 36(3): 330; 1977. (no refs.)

77-2524 Tumor Induction with Succinic Acid 2,2-Dimethylhydrazide, a Plant Growth Regulant (Meeting Abstract). (Eng.) Toth, B. (Eppley Inst. for Res. in Cancer, Univ. Nebraska, Omaha, NB) Tompa, A. *Am J Pathol* 86(2): 25a-26a; 1977. (no refs.)

77-2525 Effect of Type and Amount of Dietary Fat and 1,2-Dimethylhydrazine on Biliary Bile Acids, Fecal Bile Acids, and Neutral Sterols in Rats. (Eng.) Reddy, B. S. (Div. Nutrition, Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY 10595) Mangat, S.; Sheinfil, A.; Weisburger, J. H.; Wynder, E. L. *Cancer Res* 37(7): 2132-2137; 1977.

The effect of type (corn oil or lard) and quantity (5% or 20%) of dietary fat and 1,2-dimethylhydrazine (DMH) on the composition of biliary bile acids, fecal bile acids, and neutral sterols was studied in rats exposed to a given regimen for two generations prior to sc treatment with DMH (10 mg/kg) for 20 wk. Biliary excretion of total bile acids as well as cholic acid,  $\beta$ -muricholic acid, ursodeoxycholic acid, and deoxycholic acid was higher in rats fed diets containing 20% corn oil or lard than it was in rats fed diets containing 5% corn oil or lard. Treatment of animals with DMH increased the biliary total bile acids, cholic acid, hyodeoxycholic acid and eoxycholic acid irrespective of diets. High-fat 20% corn oil or lard intake was associated with an increased excretion of fecal neutral sterols and bile acids. The excretion of deoxycholic acid, lithocholic acid, and 12-ketolithocholic acid was increased in rats fed high-fat diets. The source of fat had no major influence on the excretory pattern of cholesterol metabolites and bile acids. DMH-treated animals excreted higher levels of fecal coprostanol, coprostanone, deoxycholic acid, lithocholic acid, and 12-ketolithocholic acid than did controls. (53 refs.)

77-2526 Metabolism of Dimethylnitrosamine and 1,2-Dimethylhydrazine in Cultured Human Bronchi. (Eng.) Harris, C. C. (Building 37, Room 3A07, NCI, NIH, Bethesda, MD 20014) Autrup, H.; Stoner, G. D.; McDowell, E. M.; Trump, B. F.; Schafer, P. *Cancer Res* 37(7):2309-2311; 1977.

Previous studies have shown that carcinogenic polynuclear aromatic hydrocarbons (PAH) are metabolically activated by

the bronchial epithelium. In the present study, dimethylnitrosamine (DMN, 27.6 or 276  $\mu$ M) and 1,2-dimethylhydrazine (1,2-DMH, 1.29 mM) bound to both cellular DNA and protein in cultured explants from three patients with lung cancer and from two patients who died from traumatic head injury. Binding of DMA to DNA showed a nonlinear dose-response relationship. The relative binding of DMA to DNA and protein varied greatly among the three patients studied: with 1,2-DMH, the interindividual variation was approx tenfold for binding to DNA and threefold for binding to protein. Liquid-column chromatography showed that bronchial DNA was methylated in the O-6 and N-7 positions of guanine. In addition to PAH, an aliphatic nitrosamine and a methylhydrazine can now be added to the list of xenobiotic chemical carcinogens metabolized by human bronchus. (33 refs.)

77-2527 An In Vitro Study on the Interaction Between Dimethylnitrosamine and Nucleic Acids via a Microsomal System. (Eng.) Grilli, S. (Istituto di Cancerologia, Universita degli Studi di Bologna, Via S. Giacomo 14, 40126 Bologna, Italy) Bragaglia, R. B.; Prodi, G. *Gann* 68(2): 129-137; 1977.

The interaction between  $^{14}$ C-dimethylnitrosamine (DMNA) and nucleic acids was studied in a microsomal system. The microsomes were extracted from the livers of rats pretreated with 3-methylcholanthrene 24 hr before sacrifice. Hydrolysates were examined by column, paper, and thin-layer chromatography. Radioactive alkylated bases were never found in the acid or alkaline hydrolysates of polyribonucleotides or in the alkaline hydrolysates of DNA after incubation. However, two radioactive compounds were always found, methylamine and N-methylhydrazine. The acid hydrolysates of DNA contained 7-methylguanine as the main interaction compound (70% of total radioactivity), with methylamine and N-methylhydrazine as minor components. The finding of 7-methylguanine as the main interaction compound in the in vitro DNA acid hydrolysates demonstrates the good agreement between in vivo and in vitro systems with respect to nucleic acid interactions. The expected methylated bases, however, were never found, giving rise to the question whether or not DMNA is really a methylating agent, as had been previously accepted. (47 refs.)

77-2528 Electron Microscopic Radioautography of Nucleic Acid Synthesis in Cultured Cells Treated with Several Carcinogens (Meeting Abstract). (Eng.) Nagata, T. (Dept. Anatomy, Shinshu Univ. Sch. Medicine, Matsumoto 390, Japan) Iwadare, N.; Murata, F. *Acta Pharmacol Toxicol [Suppl] (Kbh)* 41(1): 64-65; 1977. (7 refs.)

77-2529 The Hepatic Metabolism of  $^{15}\text{N}$  Labelled Dimethylnitrosamine in the Rat. (Eng.) Cottrell, J. C. (British Industrial Biological Res. Assoc., Woodmansterne Road, Carshalton, Surrey SM5 4DS, England) Wake, B. G.; Phillips, J. C.; Gangolli, S. D. *Biochem Pharmacol* 26(8): 809-813; 1977.

The 10,000g liver supernatant from male Sprague-Dawley rats was employed in the study of the metabolism of  $^{15}\text{N}$  labeled dimethylnitrosamine (DMN). The quantity of  $\text{N}_2$  produced during the solvolysis of hydroxy-DMN produced by the esterase-catalyzed hydrolysis of acetoxy DMN was 100% of the theoretical yield. The rates of formation of the three products of DMN metabolism, expressed as  $\mu\text{mole/g liver/hr}$  were:  $^{15}\text{N}_2$  0.114, methanol 1.88, formaldehyde, 2.42. In a similar study in the olive baboon, the rate of nitrogen production was much less than that of formaldehyde. These data, plus earlier results not consistent with the assumption that the cytochrome P-450/448 dependent mixed function oxidase system is the sole mediator of DMN metabolism, suggest that another explanation for the mechanism of nitrosamine degradation is needed. (18 refs.)

77-2530 Effect of Methylmethanesulfonate and Dimethylnitrosamine on DNA Synthesis and Thymidine Kinase Induction in Regenerating Rat Liver (Meeting Abstract). (Eng.) Gol-Winkler, R. (Laboratoire de Biochimie Appliquee, Universite de Liege, Liege, Belgium) Goutier, R. *Arch Int Physiol Biochim* 85(2): 412-413; 1977. (5 refs.)

77-2531 Effect of Dimethylnitrosamine on DNA Replication and DNA Polymerase in Liver Regenerating After Partial Hepatectomy (Meeting Abstract). (Eng.) Craddock, V. M. (MRC Toxicology Unit, Medical Res. Council Lab., Woodmansterne Road, Carshalton, Surrey SM54EF, England) *Br J Cancer* 35(2): 245; 1977. (no refs.)

77-2532 Studies on Liver Chromatin RNA from Rats Treated with N,N-Di[ $^{14}\text{C}$ ]Methylnitrosamine (Meeting Abstract). (Eng.) Galbraith, A. I. (Paterson Lab., Christie Hosp. and Holt Radium Inst., Manchester M20 9BX, England) Itzhaki, R. F. *Br J Cancer* 35(2): 248; 1977. (no refs.)

77-2533 Deuterium Isotope Effect on In Vitro Formaldehyde Formation from Dimethylnitrosamine (Meeting Abstract). (Eng.) Oshiro, Y. (NCI Frederick Can-

cer Res. Center, Frederick, MD 21701) *Proc Am Assoc Cancer Res* 18: 135; 1977. (no refs.)

77-2534 Dimethylnitrosamine Impairment of Thymidine Kinase Induction in Regenerating Rat Liver. (Eng.) Gol-Winkler, R. (Laboratoire de Biochimie Appliquee, Universite de Liege, Liege, Belgium) Goutier, R. *Arch Int Physiol Biochim* 85(1): 175-176; 1977.

Dimethylnitrosamine (9 mg/kg ip), given 30 min after partial hepatectomy, depressed the thymidine kinase activity in rat liver by 30%. When given 24 hr after surgery, it inhibited the activity only after a 24-hr delay. Increasing doses of the carcinogen progressively inhibited the enzyme when administered at 30 min, but not at 24 hr, after surgery. The inhibition might result from DNA alkylation. (4 refs.)

77-2535 Persistence of Carcinogen-Induced Initiated Hepatocytes in Liver Carcinogenesis (Meeting Abstract). (Eng.) Solt, D. (Univ. Toronto, Toronto, Ontario, Canada, M5G 1L5) Farber, E. *Proc Am Assoc Cancer Res* 18: 52; 1977. (1 ref.)

77-2536 N-Nitrosodiethanolamine in Synthetic Cutting Fluids: A Part-per-hundred Impurity. (Eng.) Fan, T. Y. (Thermo Electron Cancer Res. Center, Waltham, MA 02154) Morrison, J.; Rounbehler, D. P.; Ross, R.; Fine, D. H.; Miles, W.; Sen, N. P. *Science* 196(4285): 70-71; 1977.

A concentration of 0.02% to 3% of N-nitrosodiethanolamine was found in several brands of synthetic cutting fluids. Epidemiological studies should be performed to determine the cancer risk of this compound. (20 refs.)

77-2537 Effects of Diethylnitrosamine on Plasma Esterases and Kidney  $\beta$ -Glucuronidase in Castrated BALB/c Mice (Meeting Abstract). (Eng.) Davidson, K. A. (Biology Div., ORNL, Oak Ridge, TN 37830) Clapp, N. K.; Hall, J. W.; Congdon, C. C. *Proc Am Assoc Cancer Res* 18: 54; 1977. (no refs.)

77-2538 Temporal Advancement of Diethylnitrosamine Carcinogenesis in Aging Mice (Meeting Abstract). (Eng.) Clapp, N. K. (Biology Div., ORNL, Oak Ridge, TN 37830) Klima, W. C.; Cacheiro, L. H. *Proc Am Assoc Cancer Res* 18: 62; 1977. (no refs.)



77-2539 The Effect of Nicotinamide on the Development and Localization of Tumours, and on the Excretion of Porphyrins in the Urine of Rats Given the Hepatocarcinogen, Diethylnitrosamine (Meeting Abstract). (Eng.) Gibbard, S. (Dept. Biochemistry, Princess Alexandra Hosp., Harlow, Middlesex, England) Schoental, R. *Br J Cancer* 35(2): 254; 1977. (no refs.)

77-2540 The Utility of a Quantitative, Sensitive and Versatile Mutational Assay at the Hypoxanthine-Guanine Phosphoribosyl Transferase Locus of Chinese Hamster Ovary Cells (Cho/Hgp<sup>r</sup>t Assay) in Determining the Mutagenicity of Physical and Chemical Carcinogens (Meeting Abstract). (Eng.) Hsie, A. W. (Biological Div., Oak Ridge Natl. Lab. Oak Ridge, TN 37830) Couch, D. B.; Brimer, P. A.; O'Neill, J. P.; Machanoff, R. *Proc Am Assoc Cancer Res* 18: 160; 1977. (no refs.)

77-2541 The Identification of Chemical Carcinogens Using Rat Liver Primary Cell Cultures for the Detection of DNA Repair and Cell Mediated Mutagenesis (Meeting Abstract). (Eng.) San, R. H. (Naylor Dana Inst. Disease Prevention, Valhalla, NY 10595) Williams, G. M. *Proc Am Assoc Cancer Res* 18: 163; 1977. (1 ref.)

An Unknown Salivary Morpholine Metabolite: Identification of the Metabolite Leads to the Discovery of a New Biochemical Reaction of Secondary Amines. (Eng.) Wishnok, J. S. (Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA 02139) Tannenbaum, S. R. *Anal Chem* 49(8): 715-718; 1977.

Extracts from human saliva that had been reacted with morpholine were examined for the presence of nitrosamines by gas chromatography/mass spectrometry. In addition to N-nitrosomorpholine, the extracts contained an unidentified nitrosamine. The reactions of a variety of other amines, including diphenylamine, in the saliva yielded products with structures apparently analogous to the unknown morpholine derivative. Comparison to reference samples indicated that the derivative was a cyanamide (identical low- and high-resolution mass spectra and gas chromatography retention times). The formation of cyanamides from secondary amines in saliva is apparently fairly general, and their identification constituted the discovery of a new metabolic pathway for secondary amines. The morpholinocyanamide was found to be nonmutagenic and moderately toxic. Since most nonmutagenic compounds are noncarcinogenic, these findings indicate that the cyanamide derivatives of secondary amines are probably noncarcinogenic. The possibility that the analogous transformation of primary amines may lead to the production

of carcinogenic substances in human saliva is being investigated. (11 refs.)

77-2543 Spin Trapping and Its Application in the Study of Lipid Peroxidation and Free Radical Production with Liver Microsomes. (Eng.) Saprin, A. N. (Dept. Kinetics of Chemical and Biochemical Processes, Inst. Chemical Physics, Acad. Sciences, Moscow, USSR) Piette, L. H. *Arch Biochem Biophys* 180(2): 480-492; 1977.

Electron spin resonance was used to detect free radical adducts of phenyltertiarybutylnitrone produced during induced lipid peroxidation of microsomes with a system consisting of NADPH,  $\text{Fe}^{2+}$ , and pyrophosphate. The adducts were identified as intermediates of the substrates added to the microsomal system rather than as  $\text{OH}\cdot$  or  $\text{HO}_2\cdot$  radicals. The production of the adduct was parallel to the NADPH-dependent formation of malondialdehyde. Analyses of the electron spin resonance hyperfine splitting constants allowed identification of the adducts in some instances. Purified preparations of cytochrome P-450 mimicked the results of the microsomes. When the carcinogens dimethyl- and diethylnitrosoamine were metabolized in this system, reactive free radicals and free NO were produced, suggesting an alternate mechanism for the activity of these compounds as ultimate carcinogens. (30 refs.)

77-2544 Estrogen Therapy and Metastatic Breast Cancer (Letter to Editor). (Eng.) Buzdar, A. (Univ. Texas System Cancer Center, Houston, TX) Eckles, N. E.; Tashima, C. K. *JAMA* 237(26): 2812; 1977.

A 58-yr-old breast cancer patient who had undergone radical mastectomy was treated with diethylstilbestrol for subsequent osseous metastases. Five hr after taking the first 5 mg tablet, she was admitted with severe pain. It appears that the estrogen aggravated the metastases. (2 refs.)

77-2545 Hormone Concentration in Postmenopausal Patients with Breast Cancer. (Eng.) Jones, M. K. (Faith Courtald Unit Human Res. in Cancer, King's Coll. Hosp. and Medical Sch., London SE5, England) Ramsay, I. D.; Booth, M.; Collins, W. P. *Clin Oncol* 3(2): 177-181; 1977.

Plasma progesterone, prolactin, cortisol,  $17\beta$ -estradiol, and growth hormone concentrations were measured in 39 postmenopausal patients undergoing surgery for a breast lump. There were no significant differences in the plasma concentration of these hormones between patients with early breast cancer and those with benign breast disease. (28 refs.)

77-2546 **Hormonal Requirements for Growth In Vitro of Pregnancy-Dependent Mouse Mammary Tumors (Meeting Abstract).** (Eng.) Harbell, J. W. (Univ. California, Santa Cruz, CA 95064) Papkoff, J. S.; Daniel, C. W. *In Vitro* 13(3): 203-204; 1977. (no refs.)

77-2547 **Effect on Hypertension and Benign Breast Disease of Progestagen Component in Combined Oral Contraceptives.** (Eng.) Kay, C. R. (Royal Coll. General Practitioners, Oral Contraception Study, 8 Barlow Moor Road, Manchester M20 9RT, England) *Lancet* 1(8012): 624; 1977.

A study of combined oral contraceptives (50 µg ethinyl-estradiol with 1 mg, 3 mg, and 4 mg norethisterone acetate, respectively) indicated a negative association between benign breast disease and the dose of norethisterone acetate and a positive trend between the progestagen and hypertension. Although only three contraceptives were tested, it is believed that other combined preparations have a similar effect. (4 refs.)

77-2548 **Regression of Liver Cell Adenomas Associated with Oral Contraceptives.** (Eng.) Edmondson, H. A. (Dept. Pathology, Univ. Southern California Sch. Medicine, 2025 Zonal Ave., Los Angeles, CA 90033) Reynolds, T. B.; Henderson, B.; Benton, B. *Ann Intern Med* 86(2): 180-182; 1977.

The case histories are presented of three women who developed liver cell adenomas following use of oral contraceptives for 4, 5, and 7 yr, respectively. Cessation of steroid use resulted in tumor regression. (10 refs.)

77-2549 **Sequential Changes in the Structure and Function of Hepatocytes in Estrogen-treated *Xenopus laevis* Males.** (Eng.) Bergink, E. W. (Univ. Rochester Cancer Center, Univ. Rochester Medical Center, Rochester, NY) Tseng, M. T.; Wittliff, J. L. *Cytobiologie* 14(3): 362-377; 1977.

Alterations in the ultrastructure of hepatocytes from male frogs, *Xenopus laevis*, treated with estradiol-17β were correlated with increases in vitellogenin (VG) synthesis and secretion. Electron microscopic changes in nuclear chromatin were observed 12 hr after estrogen administration (1 mg estradiol-17β), a time when there was a two- to threefold increase in the total rate of soluble protein synthesis. An increase in VG, measured immunochemically, was barely detectable in the liver cytosol and not at all in the plasma of

treated males at 12 hr. Between 12 and 48 hr after estrogen administration, the amount of rough endoplasmic reticulum and the level of liver VG increased concurrently, indicating involvement of the total hepatocyte population in VG synthesis. <sup>3</sup>H-leucine-labeled plasma proteins from treated and untreated animals were separated on sodium dodecylsulfate-polyacrylamide gels. Almost all of the protein secreted by the liver of animals treated with estradiol 336 hr earlier was VG, but in untreated animals most of the <sup>3</sup>H-leucine was incorporated into the serum albumin. In livers of animals given multiple injections of estrogen, the rough endoplasmic reticulum occupied a large amount of the cytoplasm 336 hr after initial administration. These results suggest that estradiol-17β influences the liver of male *Xenopus* by stimulating a new differentiation of the hepatocyte through altered gene expression. (28 refs.)

77-2550 **Maternal Death Resulting from Rupture of Liver Adenoma Associated with Oral Contraceptives.** (Eng.) Kent, D. R. (Univ. California Coll. Medicine, Irvine Medical Center, 101 City Drive, South Orange, CA 92668) Nissen, E. D.; Nissen, S. E.; Chambers, C. *Obstet Gynecol [Suppl]* 50(1): 5s-6s; 1977.

A 35-yr-old woman in labor died approx 1 hr after admission. Autopsy revealed a large adenoma of the right lobe of the liver and massive hemoperitoneum. She had been taking oral contraceptives for 4 yr. (3 refs.)

77-2551 **Liver Tumors and Oral Contraceptives (Meeting Abstract).** (Ger.) Kamber, J. (Institut für Pathologie, Liestal, Switzerland) *Schweiz Med Wochenschr* 107(21): 746-747; 1977. (no refs.)

77-2552 **Adenocarcinoma of Corpus Uteri After the Use of Sequential Pill.** (Dut.) Hart, P. G. (No affiliation given) *Ned Tijdschr Geneeskde* 120(29): 1270; 1976. (no refs.)

77-2553 **Fulminant Hepatic Neoplasia After Androgen Therapy (Letter to Editor).** (Eng.) Mokrohisky, S. T. (Univ. Colorado Medical Center, Denver, CO 80262) Ambruso, D. R.; Hathaway, W. E. *N Engl J Med* 296(24): 1411-1412; 1977.

A 6-yr-old girl with no preexisting liver disease developed hepatocellular carcinoma after 2 mo of oxymethalone therapy (4.5 mg/kg/day) for Fanconi's anemia. Because of the increased incidence of neoplastic disease, even short-term ex-



posure to androgens may be contraindicated in patients with Fanconi's anemia. Bone marrow transplantation should be considered as an alternate treatment. (4 refs.)

**77-2554 Cytosol-Nuclear Binding of All Steroid Hormone Receptor Classes in the Estrogen Induced Hamster Renal Carcinoma (Meeting Abstract).** (Eng.) Li, J. J. (Res. Service, Veterans Admin. Hosp., Minneapolis, MN 5541) Cuthbertson, T. L.; Li, S. A. *Proc Am Assoc Cancer Res* 18: 141; 1977. (no refs.)

**77-2555 Endometrial Carcinoma in Women Using the Pill (Meeting Abstract).** (Dut.) Hart, P. G. (No affiliation given) *Ned Tijdschr Geneesk* 120(29): 1270; 1976. (no refs.)

**77-2556 Reduced Rate of Experimental Myosarcoma in Hormonally Bursectomized Chickens (Meeting Abstract).** (Ger.) Niedorf, H. R. (Munich, W. Germany) Lusznat, A. *Zentralbl Allg Pathol* 121(3): 296; 1977. (no refs.)

**77-2557 Effects of Prolactin and Suppression of Prolactin Secretion on Experimental Tumours of Lung and Muscle in Mice.** (Eng.) Karmali, R. A. (Clinical Res. Inst., 110 Pine Ave. W., Montreal, H2W 1R7, Canada) Horrobin, D. F. *Eur J Cancer* 13(7): 685-691; 1977.

Prolactin is a growth factor for a number of animal mammary tumors, yet it can inhibit mammary tumor development. The possibility that these actions may be systemic rather than organ-related was tested by investigating the effects of prolactin treatment and of suppression of prolactin secretion by 2-bromo- $\alpha$ -ergocryptine (bromocriptine) on two tumors of organs not thought to be prolactin-dependent. Prolactin (50 and 250  $\mu$ g, sc) appeared to inhibit the growth but not the induction of urethane-induced (1 mg/kg, ip) pulmonary adenomas in A2G mice. Bromocriptine (100  $\mu$ g, sc) had no significant actions on pulmonary adenoma growth but, when given over the period of urethane injection, it dramatically reduced the thymic uptake of tritiated thymidine. It also reduced tumor growth and increased survival in BALB/c mice injected sc with homogenates (0.2 ml) of a Moloney virus-induced rhabdomyosarcoma. Bromocriptine (250 and 500  $\mu$ g, sc) reduced survival and increased tumor growth in animals treated with the Moloney virus; prolactin given with the bromocriptine prevented these effects. These experiments suggest that the inhibitory effect of prolactin on mammary tumor growth may be partly due to the systemic effects of the hormone. The possibility that prolactin also acts on the immune system is discussed. (40 refs.)

**77-2558 Promoting Effect of Bile Acids (BA) on Colon Carcinogenesis in Germfree (GF) and Conventional (CONV) Rats (Meeting Abstract).** (Eng.) Reddy, B. S. (American Health Foundation, Valhalla, NY 10595) Watanabe, K.; Weisburger, J. H.; Wynder, E. L. *Proc Am Assoc Cancer Res* 18: 119; 1977. (no refs.)

**77-2559 The Effects of Chemical Carcinogens on New-born Epidermal Cells in Culture (Meeting Abstract).** (Eng.) Miller, D. R. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Viaje, A.; Bracken, W. M.; Slaga, T. J. *In Vitro* 13(3): 192-193; 1977. (no refs.)

**77-2560 Chemically Induced Carcinoma of Vagotomized and Resected Stomach in the Rat (Meeting Abstract).** (Ger.) Rumpf, P. (Dusseldorf, W. Germany) Schacht, U.; Palomba, P.; Schmitz, H.; Borchard, F. *Zentralbl Allg Pathol* 121(3): 297; 1977. (no refs.)

**77-2561 Formal Pathogenesis of Chemically Induced Gastric Carcinoma in the Rat After Vagotomy and Gastroenterostomy (Meeting Abstract).** (Ger.) Borchard, F. (Dusseldorf, W. Germany) Rumpf, P.; Schacht, U.; Palomba, P. *Zentralbl Allg Pathol* 121(3): 297; 1977. (no refs.)

**77-2562 Induction of Hyperplasia and Anaplasia by Carcinogens in Organ Cultures of Mouse Prostate.** (Eng.) Chopra, D. P. (Southern Res. Inst., 2000 Ninth Ave. S., Birmingham, AL 35205) Wilkoff, L. J. *In Vitro* 13(4): 260-267; 1977.

The ability of selected compounds to induce hyperplasia and anaplasia in mouse prostate organ cultures was studied to establish an in vitro system for determining the carcinogenic potential of chemicals. The compounds selected were 3-methylcholanthrene (MC), the 11-12 epoxide of MC (Ep.MC), benzo(a)pyrene (BP), pyrene (P), phenanthrene (Phe), and N-methyl-N'-nitro-N-nitrosoguanidine (MNNG). The degree of hyperplasia induced at various intervals after treatment was determined by estimating the mitotic indices using the colcemid metaphase-arrest technique. All the known carcinogens tested (MC, Ep.MC, BP, and MNNG) stimulated cell proliferation, but the noncarcinogens (P and Phe) had no effect. The mitotic stimulatory effect of the carcinogens was concentration-dependent, with max stimulation being produced at 1, 2, 4, and 8  $\mu$ g/ml of MNNG, Ep.MC, MC, and BP, respectively. Eight days after treatment, MC, Ep.MC, BP, and MNNG stimulated cellular proliferation by

332%, 141%, 441%, and 302%, respectively. At 12 days most carcinogen-treated explants were anaplastic. It is suggested that the mouse prostate organ culture system may be used as a test system to obtain preliminary information regarding the carcinogenicity of a compound. (24 refs.)

77-2563 Carcinoma of the Upper Urinary Tract Associated with Analgesic Nephropathy (Meeting Abstract). (Eng.) Storey, B. G. (Sydney Hosp., Sydney, NSW, Australia) Mahony, J. F.; Stewart, J. H. *Br J Urol* 49(3): 242; 1977. (no refs.)

77-2564 Photodynamic Action of Chlorpromazine on Adenovirus 5: Repairable Damage and Single Strand Breaks. (Eng.) Day, R. S. (Chemistry Branch, DCCP, NCI, NIH, Bethesda, MD 20014) Dimattina, M. *Chem Biol Interact* 17(1): 89-97; 1977.

Chlorpromazine ( $10^{-4}$ M), a substituted phenothiazine commonly used as a sedative, photosensitized the inactivation of human adenovirus 5 to near UV light (330-390 nanometers). The slope of the inactivation curve was threefold greater when fibroblasts from xeroderma pigmentosum (XP) patients were the viral hosts than when normal fibroblasts were used, showing that at least two-thirds of the damage produced in the virions is repairable by normal human fibroblasts. The phototreatment of chlorpromazine-sensitized virions also resulted in the production of DNA single-strand breaks, which correlated fairly well with the production of lethal viral damage in the XP fibroblasts. These findings suggest that skin photosensitization in patients treated with chlorpromazine might be due to DNA damage. (30 refs.)

77-2565 Mutagenicity for *Salmonella typhimurium* of Urine Obtained from Humans Receiving Nitrofurantoin. (Eng.) Wang, C. Y. (Div. Clinical Oncology, Dept. Human Oncology, Univ. Wisconsin Center for Health Sciences, Madison, WI 53706) Benson, R. C.; Bryan, G. T. *Natl Cancer Inst* 58(4): 871-873; 1977.

Urine samples from 12 patients receiving nitrofurantoin (100 mg po) for urinary tract infections were mutagenic for *Salmonella typhimurium* strain TA 100 and nonmutagenic for strain TA 100-FR1. Treatment with  $\beta$ -glucuronidase (2.5 mg) did not increase the mutagenicity of the urine. A spot mutation assay of thin-layer chromatograms of undiluted urine showed that the mutagenic activity was due mainly to unmetabolized nitrofurantoin. (16 refs.)

77-2566 Comparative Study of Postnatal and Transplacental Effects of Methylmethanesulfonate and Ethylmethanesulfonate (Meeting Abstract). (Ger.) Schneider, J. (Erfurt, E. Germany) Warzok, R.; Heiderstadt, R. *Zentralbl Allg Pathol* 121(3): 274; 1977. (no refs.)

77-2567 Mutagenicity Studies on Chemical Carcinogens (Meeting Abstract). (Eng.) Clive, D. (Burroughs Wellcome Co., Research Triangle Park, NC 27709) *In Vitro* 13(3): 191; 1977. (no refs.)

77-2568 Endemic Urinary Tract Cancer in Bulgaria, Yugoslavia and Roumania: Etiology and Pathogenesis. (Eng.) Markovic, B. (Medical Center, Gnjilane, Yugoslavia) Lebedev, S.; Djordjevic, M.; Arambasic, M. *Med Biol Environ* 4(1): 1-2; 1976.

The relationship between the urinary tract cancer associated with Balkan endemic nephropathy and silicate minerals in drinking water is discussed. Animal experiments indicated that nickel derived from rain-eroded quartz had the strongest carcinogenic effect because of its penetrability and affinity for the nitrogen of amino acids. (no refs.)

77-2569 Oncogenicities of the Isomeric N-Hydroxyxanthines (Meeting Abstract). (Eng.) Brown, G. B. (Sloan-Kettering Inst., New York, NY 10021) Teller, M. N.; Budinger, J. M.; Zvilichovsky, G.; Watson, A. A. *Proc Am Assoc Cancer Res* 18: 158; 1977. (no refs.)

77-2570 Induction of Rhabdomyosarcomas and Fibrosarcomas by Intratesticular Injection of Nickel Subsulfide in Rats (Meeting Abstract). (Eng.) Damjanov, I. (Univ. Connecticut Sch. Medicine, Farmington, CT 06032) Mitchell, J. M.; Allpass, P. R.; Bigazzi, P.; Sunderman, F. W. *Proc Am Assoc Cancer Res* 18: 52; 1977. (no refs.)

77-2571 The Effect of Long-term Administration of Fluorine with Food on the Carcinogenesis and Biochemical Changes in the Liver of Rats (Meeting Abstract). (Eng.) Gorban', G. P. (Moscow, USSR) Pliss, M. B.; Karpilovakaja, E. D.; Petrun', A. S.; Svatkov, V. I. *Fluoride* 10(1): 42-43; 1977. (no refs.)



- 77-2572 **Report on Carcinogenesis Bioassay of Proflavine. Availability.** (Eng.) Fredrickson, D. S. (NIH, Bethesda, MD 20014) *Fed Regist* 42(133): 35900; 1977.

The carcinogenic effects of proflavine were examined by dietary administration to mice and rats. Five of the high-dose male rats developed malignant intestinal neoplasms. The observed incidence of hepatocellular carcinoma in the high-dose, low-dose, and control groups followed a significant dose-related trend. Since there was an unusually high incidence of tumors in the male controls and since a positive-control carcinogen was tested in the same room, the validity of the findings is questioned. (no refs.)

- 77-2573 **Cell Cycle-specific Oncogenic Transformation of C3H/10T1/2 Clone 8 Mouse Embryo Cells by 1- $\beta$ -D-Arabinofuranosyleytosine.** (Eng.) Jones, P. A. (Div. Hematology-Oncology, Dept. Medicine, Childrens Hosp., Los Angeles, CA 90027) Baker, M. S.; Bertram, J. S.; Benedict, W. F. *Cancer Res* 37(7): 2214-2217; 1977.

1- $\beta$ -D-Arabinofuranosyleytosine, at concentrations ranging from  $10^{-3}$  to  $10^{-6}$  M, induces oncogenic transformation in the C3H/10T1/2 clone 8 mouse embryo cell line. Cell lines derived from type III-transformed foci grew in soft agarose and produced fibrosarcomas in immunosuppressed syngeneic mice. With cells synchronized by postconfluent inhibition of growth or isoleucine deprivation, transformation was cell-cycle-dependent. Max transformation was seen in S-phase-treated cells, although some transformation was seen in cells treated in G<sub>1</sub>. (21 refs.)

- 77-2574 **Studies on Myelofibrosis Experimentally Induced by Saponin.** (Eng.) Endo, H. (Dept. Pathology, Faculty Medicine, Univ. Tokyo, Tokyo, Japan) *Acta Haematol Jpn* 40(2): 160-171; 1977.

Myelofibrosis was induced in rabbits with saponin and the histopathological findings were compared with those of myelofibrosis in humans. Saponin was injected iv two times per week for 3 wk or for up to 9 wk. Rabbits were examined 1 or several mo after the six injections. Early changes in the marrow were extensive hemorrhage and necrosis and degeneration of hematopoietic cells due to the damaged microcirculation. As the number of injections increased, the marrow became edematous and the amount of reticulin fiber increased. Some rabbits showed thick focal fibrosis of the marrow and massive infarction. Regeneration of the hematopoietic cells was noticed after four injections. After six injections, the edema and exudation became weaker with each succeeding injection. The marrow became hypoplastic after 18 injections and, especially, 1 or 2 mo after 6 injections. Extramedul-

lary hematopoiesis, mainly in the spleen, was noticed soon after the first injection; it was more marked in the early experimental course and did not parallel the marrow fibrosis. The experimental myelofibrosis differed from the human form in that the experimental myelofibrosis was reversible, proliferation of the hematopoietic cells did not precede myelofibrosis, and splenomegaly was not found. From the electron microscopic examination, the fibroblasts were assumed to derive from reticulum cells in the marrow. The membranous structure formed at the border of the lipid droplets resembled that described as membranous lipodystrophy in 1973. (25 refs.)

- 77-2575 **Determination of Patulin in Foodstuffs. Part I: Determination of Patulin in Apple Juice.** (Ger.) Polzhofer, K. (Unilever Forschungsgesellschaft mbH, Behringstrasse 154, D-2000 Hamburg 50, W. Germany) *Z Lebensm Unters Forsch* 163(3): 183-185; 1977.

An analytical method was developed permitting the detection of 50  $\mu$ g of the carcinogen patulin per kg apple juice. After a prepurification of the crude extract by liquid-liquid extraction and column chromatography, quantitative determination of patulin resulted during remission measurements with a chromatogram-spectrophotometer at 273 nanometers. The recovery rates of additions between 120-200  $\mu$ g patulin/kg were between 82 and 90%. (13 refs.)

- 77-2576 **Cytological Effects of Insecticides on a Human Lymphoblastoid Cell Line (Meeting Abstract).** (Eng.) Trepanier, G. (Inst. Armand-Frappier, Laval-des-Rapides, Quebec, Canada H7N 4Z3) Marchessault, F.; Bansal, J.; Chagnon, A. *In Vitro* 13(3): 201; 1977. (no refs.)

- 77-2577 **Physiological Changes in Hairless Mice Maintained on an Antioxidant Supplemented Diet.** (Eng.) Chan, J. T. (Dept. Dermatology, Baylor Coll. Medicine, Houston, TX 77211) Ford, J. O.; Rudolph, A. H.; Black, H. S. *Experientia* 33(1): 41-42; 1977.

In hairless mice fed an antioxidant supplemented diet, the water soluble antioxidant content of the skin was higher than in control mice. The liver wt was increased by the antioxidant diet, possibly reflecting the enhanced ability of the liver to metabolize UV-induced carcinogens. (14 refs.)

- 77-2578 **Assay of Industrial Chemicals in Syrian Hamster Cells for Enhancement of Viral Transformation (Meeting Abstract).** (Eng.) Casto, B. C. (BioLabs,

Inc., Northbrook, IL 60062) Meyers, J.; DiPaolo, J. A. *Proc Am Assoc Cancer Res* 18: 155; 1977. (1 ref.)

77-2579 **Lead-contaminated Health Food. Association with Lead Poisoning and Leukemia.** (Eng.) Crosby, W. H. (Div. Hematology, Scripps Clinic, 1666 N. Torrey Pines Road, La Jolla, CA 92037) *JAMA* 237(24): 2627-2629; 1977.

The case history is presented of a 46-yr-old woman who developed lead poisoning and acute granulocytic leukemia while taking a prescribed bone meal calcium supplement. The lack of regulation of heavy metal content in food is emphasized. (1 ref.)

77-2580 **Induction of Morphological Transformation in Mouse C3H/10T1/2 Clone 8 Cells and Chromosomal Damage in Hamster A(T)<sub>1</sub>C1-3 Cells by Cancer Chemotherapeutic Agents.** (Eng.) Benedict, W. F. (Div. Hematology-Oncology, Dept. Medicine, Childrens Hosp., Los Angeles, CA 90027) Banerjee, A.; Gardner, A.; Jones, P. A. *Cancer Res* 37(7): 2202-2208; 1977.

Various cancer chemotherapeutic agents, including alkylating agents, antimetabolites, and antibiotics or natural products, were studied for their ability to produce morphological transformation in C3H/10T1/2 clone 8 mouse cells and chromosomal damage in A(T)<sub>1</sub>C1-3 hamster cells following 24-hr exposure of each agent at different concentrations. Those drugs known to be carcinogenic in vivo also produced morphological transformation and chromosomal damage; those agents that have not been shown to be carcinogenic in vivo produced neither transformation nor chromosomal lesions. The concentrations used in these studies were generally similar to those reached in the plasma of patients treated with these drugs for malignant and certain nonmalignant conditions. (44 refs.)

77-2581 **Leukaemia After Prolonged Use of Melphalan for Non-Malignant Disease (Letter to Editor).** (Eng.) De Bock, R. F. (Dept. Haematology, Universitaire Ziekenhuis Antwerpen, Antwerp, Belgium) Peetermans, M. E. *Lancet* 1(8023): 1208-1209; 1977.

A woman with scleromyxedema that was treated with melphalan for approx 8 yr developed leukemia at age 55, 18 mo after treatment had been stopped. Although the erythroblast triiodic acid-Schiff staining was negative, the most likely diagnosis was erythroleukemia. (4 refs.)

77-2582 **Effect of Reserpine on Cell Proliferation in the Developing Rat Brain: A Biochemical Study.** (Eng.) Patel, A. J. (Medical Res. Council Developmental Neurobiology Unit, Carshalton, Surrey, SM5 4EF, England) Bendek, G.; Balazs, R.; Lewis, P. D. *Brain Res* 129(2): 283-297; 1977.

When given to 11-day-old Porton rats at a dose of 2.5 mg/kg sc, reserpine, a well-known CNS depressant that depletes central monoamine stores, produced a severe depression in brain cell proliferation in terms of the rate of <sup>3</sup>H-thymidine incorporation into DNA. The effect was studied in detail 12 hr after administration of the drug, when the rate of in vivo DNA synthesis in the forebrain was about one-third of control. The decrease was less marked in the cerebellum (about two-thirds of control). The side effects of the drug, such as restricted food intake, hypothermia, and an elevation of blood corticosteroids were excluded from being responsible for the reduction of <sup>3</sup>H-thymidine incorporation into DNA. Kinetic studies showed that reserpine had no marked effect on the entry of <sup>3</sup>H-thymidine from blood to brain, but it caused some retardation in the rate of <sup>3</sup>H-thymidine conversion into <sup>3</sup>H-thymidine nucleotides. Nevertheless, the severe depression of DNA labeling was evident even after the values were corrected on the basis of <sup>3</sup>H-thymidine nucleotide concentrations. In contrast to these effects, thymidine kinase activity was normal in the brain of reserpine-treated animals. (53 refs.)

77-2583 **Effect of Reserpine on Cell Proliferation in the Developing Rat Brain: A Quantitative Histological Study.** (Eng.) Lewis, P. D. (Dept. Histopathology, Royal Postgraduate Medical Sch., Hammersmith Hosp., London W12 0HS, England) Patel, A. J.; Bendek, G.; Balazs, R. *Brain Res* 129(2): 299-308; 1977.

Reserpine (2.5 mg/kg body wt) depressed DNA synthesis in the forebrain of Porton rats by 2 hr postinjection; it also prolonged the cell cycle time and increased the turnover rate. In the external granular layer of the cerebellum, the mitotic index was decreased, and an increase in degenerate postmitotic nuclei was observed. (15 refs.)

77-2584 **Reactions of the Carcinogens N-Acetoxy-4-Acetamidostilbene and N-Hydroxy-4-Aminostilbene with Nucleosides (Meeting Abstract).** (Eng.) Scribner, J. D. (Pacific Northwest Res. Foundation, Seattle, WA 98104) Naimy, N. K. *Proc Am Assoc Cancer Res* 18: 131; 1977. (no refs.)

77-2585 **Metabolism of the Carcinogen O-Toluidine (Meeting Abstract).** (Eng.) Son, O. S. (Naylor Dana Inst., Valhalla, NY 10595) Weiss, L.; Fiala, E. S.; Weiss-



burger, E. K. *Proc Am Assoc Cancer Res* 18: 123; 1977. (no refs.)

77-2586 **Comparative Metabolism of Benzidine (Meeting Abstract).** (Eng.) Morton, K. C. (NCTR, Jefferson, AR 72079) King, C. M.; Baetcke, K. P. *Proc Am Assoc Cancer Res* 18: 119; 1977. (no refs.)

77-2587 **Mutagenic Activity of Styrene and Styrene Oxide. A Preliminary Study (Meeting Abstract).** (Eng.) de Meester, C. (Laboratoire de Chimie analytique, Ecole de Pharmacie, Universite de Louvain, 1200 Bruxelles, Belgium) Poncelet, F.; Roberfroid, M.; Rondelet, J.; Mercier, M. *Arch Int Physiol Biochim* 82(5): 398-399; 1977. (6 refs.)

77-2588 **Hemangiopericytoma of the Bladder after Polyvinyl Alcohol Exposure.** (Eng.) Prout, M. N. (Dept. Human Oncology, 701 C Univ. Hosp., 1300 Univ. Ave., Madison, WI 53706) Davis, H. L. *Cancer* 39(3): 1328-1330; 1977.

The case history of a 40-yr-old man who developed hemangiopericytoma of the bladder 1 yr after a 2-yr exposure to polyvinyl alcohol is presented. His exposure had consisted of daily immersion of his hand in the alcohol solution, which then hardened on his hand. Further epidemiologic and animal studies on this chemical are recommended. (11 refs.)

77-2589 **Angiosarcoma of the Liver after Vinyl Chloride Exposure: Report of a Case and Review of the Literature (Meeting Abstract).** (Eng.) Noria, D. F. (Central Hosp., Univ. Toronto, Toronto General Hosp., Toronto, Ontario, Canada) Ritchie, S.; Silver, M. D. *Lab Invest* 36(3): 361; 1977. (no refs.)

77-2590 **3-Methylindole-Induced Pulmonary Injury in Goats.** (Eng.) Huang, T. W. (Lab. Service, Veterans Admin. Hosp., 4453 Beacon Ave., Seattle, WA 98108) Carlson, J. R.; Bray, T. M.; Bradley, B. J. *Am J Pathol* 87(3): 647-667; 1977.

The pulmonary pathology in goats fed 3-methylindole was studied. The chemical caused severe pulmonary edema and respiratory distress. Electron microscopy revealed proliferation of smooth endoplasmic reticulum; thus the 3-methylindole in cigarette smoke may potentiate chemical carcinogenesis. (59 refs.)

77-2591 **Occupational Lung Cancer After Inhalation of Alkylating Compounds: Dichlorodimethyl Ether, Monochlorodimethyl Ether, and Dimethyl Sulfate.** (Eng.) Bettendorf, U. (Pathologisches Institut, der Kliniken der Landeshauptstadt, 6200 Wiesbaden, Schwalbacher Str. 62, W. Germany) *Dtsch Med Wochenschr* 102(11): 396-398; 1977.

A 42-yr-old chemist died from extensive pulmonary carcinoma after inhalation exposure to dichlorodimethyl ether, monochlorodimethyl ether, and small amounts of dimethyl sulfate over 7 yr. In experiments with rats and mice, these same chemicals led to the development of several types of cancer (fibroma, fibrosarcoma, lymphoblastic lymphosarcoma, papilloma, adenocarcinoma, neuroblastoma, ganglioneuroepithelioma, and carcinoma). It is concluded that a causal connection must exist between occupational exposure to these chemicals and carcinogenesis. (26 refs.)

77-2592 **Induction of Nasal Tumors in Rats Exposed to Hexamethylphosphoramide (HMPA) (Meeting Abstract).** (Eng.) Lee, K. P. (Haskell Lab. Toxicology and Industrial Medicine, E. I. du Pont de Nemours and Co., Inc., Wilmington, DE 19898) Trochimowicz, H. J.; Sarver, J. W. *Lab Invest* 36(3): 344-345; 1977. (no refs.)

77-2593 **Effect of BCNU, MeCCNU, DCNU, and PCNU on Developing and Fully Developed Ethylnitrosourea-induced Tumors of the Nervous System in Rats (Meeting Abstract).** (Ger.) Schiffer, D. (Halle/Saale, E. Germany) Giordana, M. T.; Pezzotta, S.; Lechner, G.; Paoletti, P. *Zentralbl Allg Pathol* 121(3): 273; 1977. (no refs.)

77-2594 **Isolation of Methylurea (MU) from Salted Dried Fish, After Nitrosation-Denitrosation (Meeting Abstract).** (Eng.) Mirvish, S. S. (Eppley Inst. Cancer Res., Univ. Nebraska Medical Center, Omaha, NB 68105) Sams, J. *Proc Am Assoc Cancer Res* 18: 165; 1977. (1 ref.)

77-2595 **Alkylation of DNA in Rat Bladder Epithelium by N-Methyl-N-Nitrosourea (MNU) (Meeting Abstract).** (Eng.) Cox, R. (Veterans Admin. Hosp. and Univ Tennessee Center for the Health Sciences, Memphis, TN 38104) Murphy, W. M.; Irving, C. C. *Proc Am Assoc Cancer Res* 18: 167; 1977. (no refs.)

- 77-2596 **The Effect of Alkylating Agents on Nuclear Poly(ADP-Ribose) Metabolism (Meeting Abstract).** (Eng.) Davies, M. I. (Biochemistry Lab., Sch. Biological Sciences, Univ. Sussex, Brighton BN1 9QC, England) Halldorsson, H.; Shall, S.; Skidmore, G. J. *Br J Cancer* 35(2): 246; 1977. (no refs.)
- 77-2597 **Effect of Ethylnitrosourea on the Activity of NAD-dependent Glycerin-3-phosphate Dehydrogenase in the N. trigeminus of Young Rats and Its Relation to Myelinization and Tumor Induction (Meeting Abstract).** (Ger.) Coutelle, R. (Berlin-Buch, E. Germany) Herrmann, V.; Schreiber, D. *Zentralbl Allg Pathol* 121(3): 275; 1977. (no refs.)
- 77-2598 **Early Stages of Experimental Brain Tumors in the Rat. Investigations of Serial Sections (Meeting Abstract).** (Ger.) Wessel, H. (Halle/Saale, E. Germany) Gerlach, H.; Schreiber, D.; Rath, F. W.; Peschel, B. *Zentralbl Allg Pathol* 121(3): 274; 1977. (no refs.)
- 77-2599 **Transplacental Carcinogenic Effect of Ethylnitrosourea in Various Animal Species (Meeting Abstract).** (Ger.) Warzok, R. (Erfurt, E. Germany) Thust, R.; Schneider, J. *Zentralbl Allg Pathol* 121(3): 273; 1977. (no refs.)
- 77-2600 **Transplacental Induction of Neurogenic Tumors in Rabbits (Meeting Abstract).** (Ger.) Stavrou, D. (Munich, W. Germany) Dahme, E.; Schroder, B. *Zentralbl Allg Pathol* 121(3): 296; 1977. (no refs.)
- 77-2601 **Direct and Transplacental Carcinogenesis by Ethylnitrosourea in the Patas Monkey (*Erythrocebus Patas*) (Meeting Abstract).** (Eng.) Rice, J. M. (NIH, Bethesda, MD 20014) London, W. T.; Palmer, A. E.; Sly, D. L.; Williams, G. M. *Proc Am Assoc Cancer Res* 18: 53; 1977. (no refs.)
- 77-2602 **Prenatal Multicarcinogenesis by Ethylnitrosourea in Mice.** (Eng.) Vesselinovitch, S. D. (Dept. Pathology, Pritzker Sch. Medicine, Univ. Chicago, Chicago, IL 60637) Koka, M.; Rao, K. V.; Mihailovich, N.; Rice, J. M. *Cancer Res* 37(6): 1822-1828; 1977.
- Pregnant mice from the cross C57BL/6J x C3HeB/FeJ and the reciprocal hybrids were given single ip injections of ethylnitrosourea (ENU) (60  $\mu$ g/g) at 12, 14, 16, and 18 days of gestation. The first-generation (F<sub>1</sub>) offspring were observed throughout their life spans. By 90 wk (av survival), they had developed primary tumors in the lungs, liver, ovaries, kidneys, nervous system, and stomach, but control mice were essentially tumor-free. Offspring of C3HeB/FeJ females and C57BL/6J males appeared to be more susceptible to lung carcinogenesis than those of the reciprocal hybrids. Fetal age at the time of ENU administration was a significant factor in the incidence of lung, liver, ovarian, and nervous system tumors. The overall incidence of liver tumors was significantly higher in males of both hybrids than in females ( $p < 0.01$ ). ENU affected a greater range of fetal tissues that that observed with polycyclic hydrocarbons or water-soluble carcinogens that require enzymatic activation. It is concluded that the sensitivity of fetal tissues to carcinogenesis is associated with stage of prenatal development of a particular tissue and, occasionally, with factors related to sex of the progeny and maternal background. (30 refs.)
- 77-2603 **Hormonal Influence on Growth and cAMP Levels in a Rat Mammary Adenocarcinoma Cell Line (Meeting Abstract).** (Eng.) Chan, P. C. (American Health Foundation, Valhalla, NY 10595) Head, J.; Tsuang, J. *In Vitro* 13(3): 190; 1977. (no refs.)
- 77-2604 **Stability Properties of Carcinogens and Procarcinogens in Cell Culture Medium (Meeting Abstract).** (Eng.) Jensen, E. M. (EG&G/Mason Res. Inst., Rockville, MD 20852) Haworth, S. R.; Kirby, P. E.; Vidrine, J. G. *Proc Am Assoc Cancer Res* 18: 118; 1977. (no refs.)
- 77-2605 **Mutagenicity and Toxicity of Various Nitrosoureas in Chinese Hamster Cells (Meeting Abstract).** (Eng.) Bradley, M. O. (Lab. Molecular Pharmacology, Div. Cancer Treatment, NCI, NIH, Bethesda, MD 20014) *Proc Am Assoc Cancer Res* 18: 60; 1977. (no refs.)
- 77-2606 **Transformation of the Fischer Rat Embryo Cell System by Two Carcinogenic Mutagens (Meeting Abstract).** (Eng.) Auletta, A. E. (Microbiological Associates, Bethesda, MD 20016) Suk, W. A. *Proc Am Assoc Cancer Res* 18: 137; 1977. (no refs.)



77-2607 Appearance of Abnormal Chromosomes Associated with Spontaneous and Chemically Induced Transformation of Cultured Chinese Hamster Cells (Meeting Abstract). (Eng.) Kirkland, D. J. (Inst. Cancer Res., Royal Marsden Hosp., Fulham Rd., London SW3 6JJ, England) Venitt, S. *Br J Cancer* 35(2): 247; 1977. (1 ref.)

77-2608 Studies on Chromosomal Aberrations and Sister Chromatid Exchanges Induced by Chemicals. (Eng.) Abe, S. (Chromosome Res. Unit, Hokkaido Univ., Sapporo, Japan) Sasaki, M. *Proc Jpn Acad* 53(1): 46-49; 1977.

Seven chemicals, N-n-butyl-N-nitrosourea, dimethylnitrosamine, 7,12-dimethylbenz[a]anthracene, 4-aminoquinoline 1-oxide, n-dibutylamine, 4-nitroquinoline 1-oxide, and sodium nitrite, were found to induce sister chromatid exchanges or chromosome aberrations in Chinese hamster cells. (6 refs.)

77-2609 Report on Carcinogenesis Bioassay of Nitrilotriacetic Acid. (Eng.) Fredrickson, D. S. (NIH, Bethesda, MD 20014) *Fed Regist* 42(96): 25534; 1977.

The carcinogenicity of nitrilotriacetic acid, trisodium salt, monohydrate was tested in both rats and mice. The compound induced urinary tract lesions in both rats and mice at the higher doses tested. These tumors were primarily of epithelial origin. (no refs.)

77-2610 On the Cellular Mechanisms of Chemical Carcinogenesis (Meeting Abstract). (Eng.) Heidelberg, C. (LAC-Univ. Southern California Cancer Center, Los Angeles, CA 90031) *Hoppe Seylers Z Physiol Chem* 358(4): 421-422; 1977. (1 ref.)

77-2611 A Technique for Evaluating Carcinogen Activation by Animal Tissues Using Hydroxylapatite Chromatography (Meeting Abstract). (Eng.) Olive, P. L. (Div. Clinical Oncology, Dept. Human Oncology, Univ. Wisconsin Center for Health Sciences, Madison, WI 53706) *Proc Am Assoc Cancer Res* 18: 60; 1977. (no refs.)

77-2612 Lysates from Hamster Embryo Cells are Selectively Cytotoxic for Chemically Transformed Cells (Meeting Abstract). (Eng.) Hatch, G. (BioLabs, Inc., Northbrook, IL 60062) Balwierz, P.; Casto, B.; Goodheart, C. *Proc Am Assoc Cancer Res* 18: 157; 1977. (no refs.)

77-2613 Behavior of Chemically Treated and nontreated Hamster Cells During Long Term Subculture In Vitro (Meeting Abstract). (Eng.) Papadopoulos, D. (Fondation Curie-Institut du Radium, 26 rue d'Ulm, 75005 Paris, France) Levy, S.; Chamaillard, L.; Hubert-Habart, M.; Saharwal, P. S.; Markovits, P. *In Vitro* 13(3): 186; 1977. (no refs.)

77-2614 In Vitro Testing for Chemical Carcinogens: Transformation Assays in Mammalian Cells (Meeting Abstract). (Eng.) Casto, B. C. (BioLabs, Inc., Northbrook, IL 60062) *In Vitro* 13(3): 191-192; 1977. (no refs.)

77-2615 DNA Repair and Mutagenesis in Mammalian Cells (Meeting Abstract). (Eng.) Fox, M. (Pater-son Lab., Christie Hosp. and Holt Radium Inst., Manchester M20 9BX, England) *Br J Cancer* 35(2): 244-245; 1977. (no refs.)

77-2616 Differential Killing Between Repair Deficient and Repair Proficient Human Cell Lines After Exposure to Carcinogens: Application As A Screening Test (Meeting Abstract). (Eng.) Burrell, A. D. (IBM Corporation, General Products Div., San Jose, CA 95193) Anderson, J. J.; Kay, E. S. *Biophys J* 17(2): 247; 1977. (no refs.)

77-2617 Comparison of DNA Repair Among Mouse Tissues and Strains in an In Vitro Assay (Meeting Abstract). (Eng.) Rasmussen, R. E. (California Coll. Medicine, Univ. California, Irvine, CA 92717) *Proc Am Assoc Cancer Res* 18: 130; 1977. (no refs.)

77-2618 Screen for Predicting Carcinogenic Potential--In Vitro DNA Damage/Alkaline Elution Assay (Meeting Abstract). (Eng.) Petzold, G. (The Upjohn Co., Kalamazoo, MI 49001) Harbach, P.; Bedell, M.; Swenberg, J. A. *Proc Am Assoc Cancer Res* 18: 63; 1977. (1 ref.)

77-2619 Mutagenicity and Antibacterial Activity of Hydroxamic Acids. (Eng.) Wang, C. Y. (Div. Clinical Oncology, Dept. Human Oncology, Univ. Wisconsin

Center for Health Sciences, Madison, WI 53706) Lee, L. H. *Antimicrob Agents Chemother* 11(4): 753-755; 1977.

The mutagenicity of various hydroxamic acids was tested against various strains of bacteria. The acids were found to have both mutagenic and antibacterial properties, probably exerted through interaction with DNA. (8 refs.)

**77-2620 9-Aminoacridine--A Frameshift Mutagen for *Salmonella typhimurium* TA 1537 Inactive at the hgp<sup>r</sup>t Locus in Human Lymphoblasts.** (Eng.) DeLuca, J. G. (Massachusetts Inst. Technology, Cambridge, MA 02139) Krolewski, J.; Skopek, T. R.; Kaden, D. A.; Thilly, W. G. *Mutat Res* 42(2): 327-330; 1977.

The frameshift mutagen of *Salmonella typhimurium* strain 1537, 9-aminoacridine, was tested for its mutagenic activity on MIT-2 human lymphoblasts derived from PGLC-33. There was no detectable activity at the hgp<sup>r</sup>t locus; the reason for this lack of activity is unknown. (7 refs.)

**77-2621 Mutagenicity of Cancer Chemotherapeutic Agents in the *Salmonella* Microsome Test.** (Eng.) Benedict, W. F. (Div. Hematology-Oncology, Dept. Medicine, Childrens Hosp., Los Angeles, CA 90027) Baker, M. S.; Haroun, L.; Choi, E.; Ames, B. N. *Cancer Res* 37(7): 2209-2213; 1977.

Seventeen cancer chemotherapeutic agents were tested for their ability to mutate *Salmonella typhimurium* tester strains in the *Salmonella*/microsome mutagenicity test. There was

a high correlation between the mutagenicity and the previously known in vivo carcinogenicity of a given agent. Carcinogens positive in the test were adriamycin, daunomycin, 1-propanol-3,3'-iminodimethanesulfonate, cyclophosphamide, isophosphamide, hycanthone, chlornaphazin, nitrogen mustard, uracil mustard, melphalan, and thio-TEPA. Two carcinogens, actinomycin D and bleomycin, were not detected as mutagens. The presumptive noncarcinogen methotrexate was negative in the test. Tilorone and 6-mercaptopurine, tentatively classified as noncarcinogens, were mutagenic. The carcinogenicity of cis-dichlorodiammineplatinum (II), which was positive in the test, has not been determined. (48 refs.)

**77-2622 On the Transfer and Expression of Prokaryotic DNA in Plant Cells Transformed by *Agrobacterium tumefaciens* (Meeting Abstract).** (Eng.) Schell, J. (Laboratorium voor Genetica, Ledeganckstraat 35, B-9000 Gent, Belgium) *Hoppe Seylers Z Physiol Chem* 358(4): 422-423; 1977. (no refs.)

\* (Rev): 77-2401, 77-2402, 77-2403, 77-2404, 77-2405, 77-2406, 77-2407, 77-2408, 77-2409, 77-2410, 77-2411, 77-2412, 77-2413, 77-2414, 77-2415, 77-2416, 77-2417, 77-2418, 77-2419, 77-2420, 77-2421, 77-2422, 77-2423, 77-2424.

\* (Phys): 77-2626, 77-2630, 77-2638, 77-2645.

\* (Viral): 77-2647, 77-2668, 77-2699.

\* (Immun): 77-2710, 77-2739, 77-2740, 77-2741, 77-2742, 77-2743, 77-2744, 77-2745, 77-2750, 77-2757, 77-2759, 77-2761, 77-2778, 77-2779, 77-2810.

\* (Path): 77-2836, 77-2843, 77-2844, 77-2852, 77-2858, 77-2871, 77-2874.

\* (Epid): 77-2883, 77-2884, 77-2885, 77-2886, 77-2887, 77-2888, 77-2900, 77-2905.



## PHYSICAL CARCINOGENESIS

- 77-2623 **Mutagenicity and Toxicity of Visible Fluorescent Light to Cultured Mammalian Cells.** (Eng.) Bradley, M. O. (Lab. Molecular Pharmacology, Div. Cancer Treatment, NCI, NIH, Bethesda, MD 20014) Sharkey, N. A. *Nature* 266(5604): 724-726; 1977.

Whether or not the toxicity and DNA strand-breaking activity of visible fluorescent light are accompanied by mutagenicity was investigated. The toxicity of fluorescent light for V-79 Chinese hamster lung cells incubated during irradiation in either complete minimal essential medium or a balanced saline soln was determined. Both survival curves were slightly shouldered, implying a saturable repair process. Medium that had been preirradiated for 3 hr in the same conditions as the cells did not reduce the colony-forming ability of the V-79 cells. Complete medium potentiated the action of light, as shown by the slightly greater killing of cells in medium compared with saline and by the decreased colony diameter and altered cell morphology caused by the preirradiated complete medium. The number of 6-thioguanine-resistant, presumably mutant colonies increased linearly as the time of exposure to cool white fluorescent light increased. After 3 hr of irradiation in complete medium, the frequency of mutants was 166x greater than that in unirradiated controls. The frequency of mutants was greater for cells irradiated in balanced saline than for cells irradiated in complete medium. Exposure to fluorescent light should be limited during any study with cultured cells. (9 refs.)

- 77-2624 **Changes of Fluoresceindiacetat-Hydrolysis and -Transport in Mammalian Cells After Ultraviolet- and Gamma Irradiation.** (Eng.) Sontag, W. (Kernforschungszentrum Karlsruhe, Institut für Strahlenbiologie, Postfach 3640, D-7500 Karlsruhe 1, W. Germany) *Radiat Environ Biophys* 14(1): 13-20; 1977.

Changes in the permeability of cell membranes and in the enzymatic activity of intact cells and their homogenates after irradiation were studied. Chinese hamster ovary cells (CHO), human RBC, and carboxylic-ester hydrolase were used as the test systems. Cells were irradiated with  $^{60}\text{Co}$   $\gamma$ -irradiation and UV light either as suspensions or as homogenates derived from the same number of cells ( $2 \times 10^6$  CHO cells/ml or  $10^8$  RBC/ml). After irradiation, 3  $\mu\text{l}$  of fluorescein diacetate (FDA) solution was added to the samples, and the amount of fluorescein produced was recorded at 37 C. Irradiation decreased the enzymatic activity in all the homogenates. Esterases were more radiosensitive in intact cells than in the corresponding homogenates. The decrease of enzymatic activity after irradiation can be described by a one-hit curve;

the loss of transport activity can be resolved into two one-hit curves. Mechanisms for the decrease in the rate constants are discussed. It is concluded that intracellular FDA hydrolysis is an excellent tracer method for studying the interaction between radiation and intracellular esterase and transport activity. (14 refs.)

- 77-2625 **The Study of Remote Sequelae of Prolonged Experimental Application of Long-Wave Ultraviolet Radiation.** (Rus.) Dantsig, N. M. (A. N. Sysin Inst. General and Communal Hygiene, Acad. Medical Sciences USSR, Moscow, USSR) Prokopenko, Iu. I.; Zabalueva, A. P. *Vestn Akad Med Nauk SSSR* (2): 42-46; 1977.

Long-term exposures to suberythematous doses of long-wave UV radiation accelerated the aging process in mice and had a teratogenic effect on the offspring. These findings suggest the possibility of the carcinogenic effect of high UV doses. (9 refs.)

- 77-2626 **Effect of Fluorescent Light and Oxygen on Chromosomes, Cytology, and Neoplastic Transformation of Mouse Cells in Culture (Meeting Abstract).** (Eng.) Sanford, K. K. (Lab. Biochemistry, NCI, Bethesda, MD 20014) Parshad, R.; Handleman, S. L.; Jones, G. M.; Price, F. M. *In Vitro* 13(3): 181; 1977. (no refs.)

- 77-2627 **Cellular and Tissue Changes in Mouse Thyroid After Local X-Ray Irradiation.** (Eng.) Kashtenko, L. A. (Inst. Morphology, Bulgarian Acad. Sciences, Sofia, Bulgaria) Bakalska, M. V. *C R Acad Bulg Sci* 30(1): 141-144; 1977.

Morphological changes in the thyroid gland of mice following various doses of x-irradiation were studied. The size of the induced lesions was directly proportional to the dose. Irradiation first stimulated the gland; then a decrease in activity followed. (7 refs.)

- 77-2628 **Alpha Particle Dose to Respiratory Airway Epithelium.** (Eng.) Desrosiers, A. E. (Dept. Physi-

ology, Harvard Sch. Public Health, 665 Huntington Ave., Boston, MA 02115) *Health Phys* 32(3): 192-195; 1977.

The use of the Haque and Nelson equations for calculating the hazard of alpha particle dose to the respiratory epithelium results in an overestimation of the depth dose distribution. A third set of calculations were carried out to obtain more accurate values; these results were in agreement with other estimates of depth dose in airways. (12 refs.)

77-2629 **Alpha-particle-induced Transformation in a C3H Mouse-Embryo-Derived Cell Line (Meeting Abstract).** (Eng.) Lloyd, E. L. (Argonne Natl. Lab., Argonne, IL 60439) Gemmell, A.; Henning, C. B.; Gemmell, D. S.; Zabransky, B. J. *In Vitro* 13(3): 181; 1977. (no refs.)

77-2630 **Influence of Steroid Hormones on the Carcinogenicity of <sup>90</sup>Sr.** (Eng.) Nilsson, A. (Res. Inst. Natl. Defence, S-172 04 Sundbyberg, Sweden) Broome-Karlsson, A. *Acta Radiol [Ther] (Stockh)* 15(5): 417-426; 1976.

The effects of estrogens and steroid hormones on the carcinogenicity of <sup>90</sup>Sr were investigated in female CBA mice. The mice were given <sup>90</sup>Sr ip as <sup>90</sup>Sr(NO<sub>3</sub>)<sub>2</sub>, alone or in combination with polyestradiophosphate, methylprednisolone or nortestosterone sc. When <sup>90</sup>Sr was given in combination with polyestradiophosphate, the frequency of osteosarcomas was significantly increased and the tumor latency time was decreased, compared to mice given <sup>90</sup>Sr alone. These effects were not seen with <sup>90</sup>Sr in combination with nortestosterone. The combination of <sup>90</sup>Sr and methylprednisolone resulted in a great reduction of the osteosarcoma incidence and a prolonged tumor latency time. It had been previously suggested that estrogenic hormones exert their promoting action on <sup>90</sup>Sr carcinogenicity by stimulating the irradiated bone cells to proliferation. This would mean that <sup>90</sup>Sr irradiates not only a much more numerous cell population but also a constantly stimulated one. This indicates that overpopulation is the most decisive factor, since the greater the population irradiated, the greater the chance for malignant clones to develop. This theory also seems to fit the results obtained in the corticosteroid series, in which the irradiated bone cells were influenced by an inhibitory factor. Previous reports had indicated that corticosteroid hormones given continuously for a long period induce a decrease in the activity of bone cells concomitantly with a reduction of their numbers. As a consequence, the bone tissue is devoid of much of its reparative response and proliferative capacity. This is probably the main reason why the tumor frequency is diminished in animals given methylprednisolone. (17 refs.)

77-2631 **The Morphology of Lung Tumors Induced in Rats with Inhalation of Plutonium Nitrate (Meeting Abstract).** (Eng.) Dagle, G. E. (Battelle Northwest Lab., Richland, WA 99352) McDonald, K. E.; Ballou, J. E. *Lab Invest* 36(3): 335; 1977. (no refs.)

77-2632 **DNA Repair in *Bacillus subtilis*. I. The Presence of an Inducible System.** (Eng.) Yasbin, R. E. (Dept. Microbiology, S231 Frear Building, Pennsylvania State Univ., University Park, PA 16802) *Mol Gen Genet* 153(2): 211-218; 1977.

The inducible DNA repair system following UV irradiation of *Bacillus subtilis* was investigated and compared to the *Escherichia coli* 'SOS' system, in which DNA damage results in the release of a signal which simultaneously activates mechanisms to aid cell survival. (49 refs.)

77-2633 **Expression of an Excision Repair Gene in Transformation of *Bacillus subtilis*.** (Eng.) Tanooka, H. (Radiobiology Div., Natl. Cancer Center Res. Inst., Tsukiji, Tokyo 104, Japan) Takahashi, A. *Mol Gen Genet* 153(2): 129-133; 1977.

The effect of a donor repair gene in a strain of *Bacillus subtilis* that was deficient in excision repair was investigated. The donor repair gene was found to affect the UV survival curve of the cells; this resulted in a UV-resistant component. (14 refs.)

77-2634 **DNA Replication and Repair in a Human Melanoma Cell-Line Resistant to Ultra-Violet-Radiation.** (Eng.) Lavin, M. F. (Dept. Biochemistry, Univ. Queensland, Brisbane, 4067, Australia) Willett, G. M.; Chalmers, A. H.; Kidson, C. *Int J Radiat Biol* 31(2): 101-111; 1977.

A report is presented on the effect of UV irradiation on semi-conservative synthesis of DNA and on repair functions associated with DNA replication in a UV-resistant, human melanoma cell line (MM96). Cells were double-labeled with <sup>14</sup>C and <sup>3</sup>H thymidine. Semiconservative synthesis of DNA was decreased about fivefold by a UV dose of 100 ergs/mm<sup>2</sup>. The molecular wt of DNA fragments synthesized in irradiated cells at short times after UV were smaller than those synthesized in unirradiated cells. Elongation of these fragments occurred with time, and 6 hr after irradiation, cells synthesized DNA in fragments of the same size as those of unirradiated cells. In this



post-replication repair process, elongation appeared to involve new synthesis and was not inhibited by theophylline. (26 refs.)

- 77-2635 **Contribution to the Study of Excision Repair Following UV Irradiation in Mammalian Cells Cultivated In Vitro.** (Eng.) Hochmann, J. (Cancer Res. Inst., Slovak Acad. Sciences, 880 32 Bratislava, Czechoslovakia) Masek, F.; Chalupa, I. *Neoplasma* 23: 623-628; 1976.

Cultures of embryonal Lewis White rat LEF cells in their 126th passage and HeLa cells were labeled with 10 and 5  $\mu$ Ci/ml thymidine-6-<sup>3</sup>H, respectively. After 24 hr, cells were irradiated with given doses of UV light and removed from culture at indicated time intervals to determine the presence of thymidine dimers. Using either enzymatic (lysozyme-pronase) or chemical (chloroformisoamyl alcohol) deproteinization methods, DNA was isolated, and the content of TT (tritiated thymidine) was estimated radiochromatographically. At UV doses of 75 and 300 ergs/mm<sup>2</sup>, the LEF cells were unable to excise thymine dimers even after 48 hr. HeLa cells irradiated with 300 ergs/mm<sup>2</sup> showed evident excision of TT in normal medium after 12 hr; however, excision was depressed in cultivation medium acidified with H<sub>3</sub>PO<sub>4</sub> (pH 5.83). The two different methods of deproteinization gave the same results. The pH of the extracellular environment may influence excision repair. (20 refs.)

- 77-2636 **Recombination of UV-Induced Pyrimidine Dimers in Human Fibroblasts (Meeting Abstract).** (Eng.) Waters, R. (Biology Div. Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Regan, J. D. *Biophys J* 17(2): 141; 1977. (no refs.)

- 77-2637 **Differences in Strand Break Repair Between Fibroblasts from Different Species (Meeting Abstract).** (Eng.) Macieira-Coelho, A. (Institut de Cancerologie et Immunogenetique, INSERM-U50, 94800, Villejuif, France) Diatloff, C.; Loria, E. *In Vitro* 13(3): 186; 1977. (no refs.)

- 77-2638 **Postreplication Repair in Xeroderma Pigmentosum Fibroblast Cells (Meeting Abstract).** (Eng.) Minka, D. F. (West Virginia Univ., Morgantown, WV 26506) Nath, J. *In Vitro* 13(3): 201; 1977. (no refs.)

- 77-2639 **Localization of Inhibition of Replicon Initiation to Damaged Regions of DNA in Cultured Mammalian Cells (Meeting Abstract).** (Eng.) Povirk, L. F. (Lab. Radiobiology, Univ. California, San Francisco, CA 94143) Painter, R. B. *Biophys J* 17(2): 143; 1977. (no refs.)

- 77-2640 **DNA Repair in Arrested Human Diploid Fibroblast Cultures Irradiated with Ultraviolet Light (Meeting Abstract).** (Eng.) Kantor, G. J. (Dept. Biological Sciences, Wright State Univ., Dayton, OH 45431) Hull, D. R. *Biophys J* 17(2): 144; 1977. (no refs.)

- 77-2641 **DNA Repair in Ultraviolet Irradiated Normal and Malignantly Transformed Mouse Epidermal Cell Cultures (Meeting Abstract).** (Eng.) Bowden, G. T. (NIH, Bethesda, MD 20014) Fusenig, N. E. *Proc Am Assoc Cancer Res* 18: 145; 1977. (no refs.)

- 77-2642 **Damage and Repair of a Non-Freely-Sedimenting DNA Component Following Low and High LET Irradiation and Hyperthermic Pretreatment of CHO Cells (Meeting Abstract).** (Eng.) Cole, A. (Physics Dept., Univ. Texas System Cancer Center, Houston, TX 77030) Kristal, I. *Biophys J* 17(2): 143; 1977. (no refs.)

- 77-2643 **Comments on Yerushalmi's Article (Reference 1) (Letter to Editor).** (Eng.) Atkinson, E. R. (NCI, Div. Cancer Treatment, Bethesda, MD 20014) *Eur J Cancer* 13(2): 193-194; 1977.

Investigations are in progress to determine whether prolonged exposure to temperatures less than 41.5 C leads to increased tumor proliferation in vivo during treatment with local and whole body hyperthermia. It is suggested that the lowest rather than the highest temperature attained by the cells during treatment would be the best measurement of effectiveness. (5 refs.)

- 77-2644 **DNA Damage and "Repair" in V79 Cells Irradiated as Monolayers or Multicell Spheroids**

(Meeting Abstract). (Eng.) Durand, R. E. (Div. Radiation Oncology, Wisconsin Clinical Cancer Center, Univ. Wisconsin Medical Sch., Madison, WI 53706) Olive, P. L. *Biophys J* 17(2): 244; 1977. (no refs.)

77-2645 **Contribution of Secondary Radicals to Indirect Action of Radiation (Meeting Abstract).** (Eng.) Becker, D. (Dept. Physics, Illinois Inst. Technology, Chicago, IL 60616) Grossweiner, L. I. *Biophys J* 17(2): 144; 1977. (1 ref.)

77-2646 **Search for an Inducible System Responsible for Mutagenesis in Mammalian Cells (Meeting Abstract).** (Eng.) Cleaver, J. E. (Lab. Radiobiology, Univ. California, San Francisco, CA 94143) *Biophys J* 17(2): 291; 1977. (no refs.)

\* (Rev): 77-2423, 77-2424, 77-2425, 77-2426, 77-2427, 77-2428, 77-2429, 77-2430, 77-2431.

\* (Chem): 77-2449, 77-2499, 77-2540, 77-2564, 77-2577.

\* (Immun): 77-2730, 77-2756, 77-2781.

\* (Path): 77-2867.

\* (Epid): 77-2889, 77-2890, 77-2891.



## VIRAL CARCINOGENESIS

- 77-2647 **Comparative Studies on Isoaccepting Arginyl tRNAs from Transformed Cells and Their Utilization in Post-translational Protein Modification.** (Eng.) Rao, P. (Dept. Pathology, Univ. Toronto, Toronto, Canada) Kaji, H. *Arch Biochem Biophys* 181(2): 591-595; 1977.

The effect of viral infection and transformation of cells on the isoaccepting transfer RNA's (tRNA's) for arginine and their utilization in this protein modification reaction were investigated. Posttranslational arginine-incorporating activity was studied in polyoma-infected baby mouse kidney (BMK) cells, simian virus 40 (SV40)-transformed 3T3 cells, rat embryo cells transformed by dimethylbenzanthracene (DMBA) and 2-methylcholanthrene (MC), and their normal counterparts. The incorporation of <sup>3</sup>H-arginine into hot trichloroacetic acid-insoluble radioactivity was measured by the filter paper disk method. The results showed that transfer of arginine from arginyl-tRNA (arg-tRNA) was better with the extract obtained from polyoma-infected BMK cells than extracts from uninfected controls. Similar efficiency of transfer was observed between the extracts of SV40-3T3 and control 3T3 cells. Extracts of rat embryo cells transformed by DMBA, but not by MC, had a higher arginine-incorporating activity than control cells. The chromatographic properties of isoaccepting arg-tRNA's derived from transformed or infected cells and their controls were similar on RPC-5 columns. The transfer of arginine from two species of isoaccepting arg-tRNA's of polyoma-infected BMK cells was better than that from arg-tRNA's of uninfected controls. It is concluded that there is no consistent quantitative or qualitative difference in arginine incorporation between control and transformed/infected cell types. (24 refs.)

- 77-2648 **The Oncornavirus Maturation Process: Quantitative Correlation Between Morphological Changes and Conversion of Genomic Virion RNA.** (Eng.) Korb, J. (Dept. Molecular Virology, Inst. Molecular Genetics, Czechoslovak Acad. Sciences, Flemingovo namesti 2, Prague 6, Czechoslovakia) Travnicek, M.; Riman, J. *Inter-virology* 7(4/5): 211-224; 1976.

Avian myeloblastosis virus (AMV) was harvested at different time intervals from chick leukemic myeloblasts, and the rate of virus maturation was estimated on the basis of morphological changes in the virions and conversion of genomic viral RNA. The change from immature virions, characterized by the presence of an electron-lucent center, to the condensed form (with a dense nucleoid) was accompanied by conversion of 30S-40S RNA to 60S RNA. Both processes were quantitatively defined and correlated and found to proceed in parallel

at the same rate. Early stages of maturation were characterized by a high turnover of immature to mature virions; 31%-40% of the mature forms were present in 3.5-min virus harvests. The rate of this process decreased with time, although the 17-hr harvests still contained 10%-15% of immature virions. The course of maturation supports the hypothesis that the transformation of A particles to C particles is not a simple matter of passive aging, but involves specific active processes. The decrease in maturation with time may be due to the degradation of some time-labile factor needed for extracellular maturation. Alternatively, some viral particles may be poorly assembled and unable to undergo maturation after their release. (25 refs.)

- 77-2649 **Terminally Repeated Sequences in the Avian Sarcoma Virus RNA Genome.** (Eng.) Collett, M. S. (Dept. Microbiology, Univ. Minnesota Medical Sch., Minneapolis, MN 55455) Dierks, P.; Cahill, J. F.; Faras, A. J.; Parsons, J. T. *Proc Natl Acad Sci USA* 74(6): 2389-2393; 1977.

Based on studies of B77 virus, the initiation of DNA synthesis in vitro by the RNA-directed DNA polymerase of avian oncornaviruses was found to require a transfer RNA primer molecule (tRNA-trp) located close to the 5' end of the viral RNA genome. DNA transcripts, 100 nucleotides in length, initiated on the tRNA-trp primer molecule contain nucleotide sequences complementary to a large (25 nucleotides) RNase T<sub>1</sub> oligonucleotide, T-13, located at the 5' terminus of the avian sarcoma virus (ASV) RNA genome. tRNA-trp-initiated DNA transcripts with a length of about 70 nucleotides contain substantially fewer nucleotide sequences complementary to this 5'-terminal oligonucleotide, suggesting that the tRNA-trp primer associated with the ASV RNA is located approx 100 nucleotides from the 5' end of the RNA. DNA transcribed from ASV RNA sequences located at the 3' end, immediately adjacent to the poly(A), contains nucleotide sequences that are complementary to the 5'-terminal T<sub>1</sub> oligonucleotide T-13. These data indicate that the 5' end of the viral genome contains nucleotide sequences that are repeated at the 3' end of the genome. The terminally redundant nature of the virus RNA genome is discussed in relation to the mechanism by which the genome is converted into the circular form of DNA. (25 refs.)

- 77-2650 **Characterization of DNA Complementary to Nucleotide Sequences at the 5'-Terminus of the Avian Sarcoma Virus Genome.** (Eng.) Friedrich, R. (Max-

Planck-Inst. Molecular Genetics, Ihnestrasse 63-73, D-1000 Berlin 33, W. Germany) Kung, H. J.; Baker, B.; Varmus, H. E.; Goodman, H. M.; Bishop, J. M. *Virology* 79(1): 198-215; 1977.

The 5'-end of the genome of avian sarcoma virus (ASV) contains a nucleotide sequence that is the initial template for transcription by RNA-directed DNA polymerase. DNA complementary to this sequence (cDNA) was prepared and used to characterize the sequence by chemical analysis and molecular hybridization. The length of cDNA<sub>5</sub> (100 nucleotides) provides an estimate of the distance from the primer for DNA synthesis (tRNA-trp) to the 5'-terminus of the genome. As previously reported, transcription of DNA from the genome of ASV in vitro can be arrested at preferred sites short of the 5'-terminus by reducing the concentration of either deoxycytidine triphosphate or deoxyguanosine monophosphate. The nucleotide sequence complementary to cDNA is similar but not identical in the genomes of two strains of ASV, and homologous sequences are present in the genomes of other avian leukosis-sarcoma viruses. By contrast, there is little or no complementarity between cDNA<sub>5</sub> and the genomes of golden pheasant virus (subgroup G of avian leukosis-sarcoma virus), Moloney murine leukemia virus, and termini of RNA tumor virus genomes. A nucleotide sequence complementary to most or all of cDNA occurs within the 3'-half, but not at the 3'-terminus, of the ASV genome; this sequence may be a ribosome-binding site. cDNA constitutes the bulk (usually > 75%) of DNA synthesized under conventional conditions with detergent-activated virions. Consequently, the use of this DNA to analyze homologies among retrovirus genomes is of limited value. (43 refs.)

77-2651 **Localization of N<sup>6</sup>-Methyladenosine in the Rous Sarcoma Virus Genome.** (Eng.) Beemon, K. (Tumor Virology Lab., Salk Inst., Post Office Box 1809, San Diego, CA 92112) Keith, J. *J Mol Biol* 113(1): 165-179; 1977.

The 10,000-nucleotide RNA genome of the Prague strain, subgroup B (PR-B) of Rous sarcoma virus (RSV) was found to contain 11.6 residues of N<sup>6</sup>-methyladenylic acid (m<sup>6</sup>Ap) by quantitative analysis of <sup>32</sup>P-labeled virion RNA after complete RNAase digestion. Approximately 10 of the m<sup>6</sup>Ap residues are located, without obvious clustering, in that region of the genome between 500 and 4,000 nucleotides from the 3' poly(A) end. The src gene, which is required for transformation, and part of the env gene, which codes for the major viral envelope glycoprotein, have previously been mapped in this region of the viral genome. A transformation-defective deletion mutant of PR-B RSV, which lacks the src gene, has 7.0 m<sup>6</sup>Ap residues per RNA subunit. This supports the mapping of a portion of the N<sup>6</sup>-methyladenosine (m<sup>6</sup>A) residues in src and suggests that this methylation is specific to certain

regions of the genome. The possible significance of this result for RSV RNA processing and translation is discussed. (49 refs.)

77-2652 **The Effect of Induced Membrane Crystallinity on Rous Sarcoma Virus-transformed Chicken Embryo Fibroblasts (Meeting Abstract).** (Eng.) Baker, P. E. (Univ. Connecticut, Storrs, CT 06268) *Diss Abstr Int [B]* 38(1): 29; 1977. (no refs.)

77-2653 **Interaction of Oncornaviruses with Host Genomes (Meeting Abstract).** (Eng.) Humphries, E. (Imperial Cancer Res. Fund Lab., Lincoln's Inn Fields, London WC 2A 3 PX, England) Weiss, R. *Hoppe Seylers Z Physiol Chem* 358(4): 421; 1977. (no refs.)

77-2654 **Alterations of Neoplastic Characteristics of a Glutamine-independent Variant (GIV) of Polyoma-BHK (Meeting Abstract).** (Eng.) Gammon, M. T. (Massachusetts General Hosp., Boston, MA 02114) Isselbacher, K. J. *In Vitro* 13(3): 172; 1977. (no refs.)

77-2655 **Invasive Metastasizing Carcinomas from Embryonic Salivary Epithelium Infected with Polyoma Virus In Vitro (Meeting Abstract).** (Eng.) Dawe, C. J. (NCI, Bethesda, MD 20014) Morgan, W. D.; Williams, J. E.; Summerour, J. P. *Proc Am Assoc Cancer Res* 18: 164; 1977. (no refs.)

77-2656 **Synthesis and Cleavage Processing of Oncornavirus Proteins During Interferon Inhibition of Virus Particle Release.** (Eng.) Shapiro, S. Z. (Dept. Molecular Biology, Albert Einstein Coll. Medicine, Bronx, New York, NY 10461) Strand, M.; Billiau, A. *Infect Immun* 16(3): 742-747; 1977.

The effect of interferon on synthesis rate and cleavage processing of viral proteins in mouse cells (NIH/3T3) chronically infected with Rauscher murine leukemia virus was studied by immunoprecipitation of newly synthesized viral proteins from virus-infected cells pulse-labeled with <sup>35</sup>S-methionine. Immunoprecipitated, labeled polypeptides were resolved by polyacrylamide gel electrophoresis in the presence of sodium dodecyl sulfate and then examined by autoradiography. Cleavage processing was studied in the same manner with cells that had been pulse-labeled and then incubated with



nonradioactive media for a sufficient time to allow normal cleavage processing to occur. At a concentration (1,000 reference units/ml) that strongly inhibited the release of virus particles, interferon had no effect on the synthesis of proteins carrying antigenic determinants of the major core protein P30 or of the envelope glycoprotein gp69/71; nor did it affect the posttranslational cleavage processing of the precursors to these proteins. Similarly, interferon did not affect the labeling or chasing of precursor protein carrying the p15 determinants; labeling of p15 itself could not be studied because it does not contain methionine. (22 refs.)

- 77-2657 **Bromodeoxyuridine Inhibition of Friend Leukemia Cell Induction: Mechanism of Reversal by Deoxycytidine.** (Eng.) Bick, M. D. (Roche Inst. Molecular Biology, Nutley, NJ) *Biochim Biophys Acta* 476(4): 279-286; 1977.

The mechanism by which deoxycytidine (dC) reverses bromodeoxyuridine (BUdR) inhibition of erythroid differentiation in Friend leukemia cells was studied. Cultures were labeled with <sup>3</sup>H-BUdR with or without dC. Butyric acid was also added at zero time, and samples were withdrawn and separated into methanol-soluble and -insoluble fractions at various intervals thereafter. The methanol-insoluble fraction was used to determine precipitable radioactivity and to establish radioactivity in DNA. Soluble nucleotide pools were used for separation of the nucleosides and the mono-, di-, and triphosphates of BUdR. It was found that dC reverses BUdR inhibition of induction only if added within the first 6 hr after BUdR. dC reduced the uptake of <sup>3</sup>H-BUdR into both soluble nucleotide pools and DNA, substantially expanded the deoxythymidine 5'triphosphate pool, and resulted in a lower level of BUdR substitution in DNA. When the conversion of dC to thymidine nucleotides was prevented in BATH medium (containing BUdR, aminopterin, thymidine, and hypoxanthine), dC no longer reversed BUdR inhibition. These results show that dC exerts its primary effect via alterations in thymidine pools and probably through the resultant lower substitution of BUdR in DNA. (15 refs.)

- 77-2658 **Purification and Analysis of a Gross Murine Oncornavirus Protein with a Molecular Weight of About 12,000 Specific for the Gross Virus Subgroup.** (Eng.) Strand, M. (Dept. Pharmacology and Experimental Therapeutics, Johns Hopkins Univ. Sch. Medicine, 725 N. Wolfe St., Baltimore, MD 21205) August, J. T. *Virology* 79(1): 129-143; 1977.

A structural protein with a molecular wt (MW) of approx 12,000 was purified from Gross murine oncornavirus by phosphocellulose column chromatography. The protein was distinct from the other principal Gross virus components and did not react with antisera prepared against purified Gross

virus proteins with MW's of about 30,000, 17,000, 14,000, and 10,000. The immunologic properties of the protein were analyzed by competition radioimmunoassay. The antigenic determinants were chiefly subgroup-specific, with a close homology to the analogous protein of AKR, Kirsten, or BALB/c xenotropic viruses but not to that of Friend, Rauscher, Moloney, NIH Swiss xenotropic, or NAB xenotropic viruses. Group-specific determinants cross-reactive with the analogous protein of these latter viruses were present but were minor components of the total antigenic specificities of the protein. Weak interspecies cross-reactivity was demonstrated with the Theilen feline virus, but very little, if any, with different primate oncornaviruses. The identity of the related, cross-reactive proteins of other murine viruses was analyzed by competition radioimmunoassay and immunoprecipitation. They were identified as the 15,000-MW component of Rauscher and Friend viruses and the 12,000-MW component of Moloney and BALB/c xenotropic viruses. The close similarity in the properties of the p15 of Rauscher murine virus and the p12 of Gross murine virus establishes them as the homologous subgroup-specific proteins of the FMR and Gross subgroups of viruses. (46 refs.)

- 77-2659 **Mechanism of Fv-1 Locus Inhibition of Murine Leukemia Viruses: Studies by DNA Transfection (Meeting Abstract).** (Eng.) Yang, W. K. (Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Hsu, I. C.; Boone, L. R.; Tennant, R. W.; Brown, A. *Proc Am Assoc Cancer Res* 18: 149; 1977. (no refs.)

- 77-2660 **Preleukemia in the Rat: Comparative Study of Virus-Induced Changes (Meeting Abstract).** (Eng.) Siegler, R. (Drew Postgraduate Medical Sch. and Martin Luther King, Jr. Hosp., Los Angeles, CA 90059) *Proc Am Assoc Cancer Res* 18: 138; 1977. (no refs.)

- 77-2661 **Effect of Paul. Bunnell (PB) Antigen on the Spleen Focus Assay for Murine Leukemia Viruses Using Polycythemic Friend Virus (PFV) (Meeting Abstract).** (Eng.) Fjelde, A. (Roswell Park Memorial Inst., Buffalo, NY 14263) Evege, E. *In Vitro* 13(3): 171-172; 1977. (no refs.)

- 77-2662 **Inherited Resistance to N- and B-Tropic Murine Leukemia Viruses In Vitro. Effect of the Fv-1 Locus on the Rescue of a Replication-defective and Transformation-defective Murine Sarcoma Virus in the Fv-1 Congenic Strains SIM.S and SIM.R.** (Eng.) Kochman, M. A. (Dept. Anatomy, Div. Histology, Medical Sciences Build-

ing, Univ. Toronto, Toronto, Ontario, Canada M5S 1A8) Blackstein, M. E.; McCarter, J. A. *Virology* 79(2): 302-311; 1977.

An in vitro assay for murine leukemia virus (MuLV) using mouse embryo cultures derived from the congenic strains SIM.S and SIM.R differing at the Fv-1 locus and containing the genome of a replication- and transformation-defective (R-T-) murine sarcoma virus isolate is described. Studies of the interrelationship of MuLV and MuSV, performed with this focus induction assay, indicated that rescue of the defective MuSV was subject to Fv-1 restriction of the superinfecting MuLV helper. Titration patterns of N-, B-, and NB-tropic MuLV on these SIM.S and SIM.R R-T- cells were all linear and parallel over the range of dilutions used. The kinetics of MuLV infection appeared to be single-hit on the basis of focus and XC-plaque assays in both permissive and nonpermissive host cells. MuSV rescued by N-, B-, or NB-tropic MuLV showed no strict host range specificity. The host range of these rescued MuSV was determined by the degree of restriction of coinfecting MuLV. The results with both normal and MuSV(R-T-)-infected SIM.S(Fv-1n/n) and SIM.R(Fv-1b/b) cell lines suggest that Fv-1-mediated restriction acts solely by restricting MuLV replication and thereby inhibiting focus formation by the defectivestive MuSV genome in Fv-1 nonpermissive cells. (29 refs.)

77-2663    **The Major Polypeptides of the Murine-Mammary-Tumor Virus Isolated by Plant-Lectin Affinity Chromatography.** (Eng.) Westenbrink, F. (Radiobiological Inst., Nederlandsche Organisatie voor Toegepast-Natuurwetenschappelijk Onderzoek, Lange Kleiweg 151, Rijswijk, Netherlands) Koornstra, W.; Bentvelzen, P. *Eur J Biochem* 76(1): 85-90; 1977.

Solubilized polypeptides of the murine mammary tumor virus isolated from BALB/cfC3H mammary tumors were chromatographed on a column of immobilized concanavalin A. The unbound viral material was rechromatographed on phosphocellulose, resulting in isolation of the major internal proteins with a molecular wt of 28,000 (p28) and 12,000 (p12), respectively. After elution with methyl  $\alpha$ -D-mannopyranoside, the adsorbed glycopolypeptides were subjected to gel filtration. The major glycoprotein, with a molecular wt of 52,000 (gp52), was obtained in almost pure form. However, a considerable part of gp52 eluted together with a glycoprotein with a molecular wt of 36,000 (gp36), suggesting that gp52 occurs as a complex with gp36 as well as in the free form. (22 refs.)

77-2664    **Nutritional Factors That Influence Expression of Mouse Mammary Tumor Virus: Iron, Zinc and Lipids (Meeting Abstract).** (Eng.) Nagle, S. C. (NCI Frederick Cancer Res. Center, P.O. Box B, Frederick, MD

21701) Fine, D. L.; Kmetz, J. P. *In Vitro* 13(3): 173; 1977. (1 ref.)

77-2665    **Localization of Mouse Mammary Tumor Virus Antigen on Intracytoplasmic A Particle Structural Polypeptides (Meeting Abstract).** (Eng.) Smith, G. H. (Lab. Molecular Biology, NCI, Bethesda, MD 20014) *Proc Am Assoc Cancer Res* 18: 126; 1977. (no refs.)

77-2666    **RNA-dependent DNA Polymerase Activity in Intracytoplasmic Type-A Particles of Mouse Mammary Tumors.** (Eng.) Wunderlich, V. (Zentralinstitut für Krebsforschung der Akademie der Wissenschaften der DDR, Bereich Tumorstudiologie, 1115 Berlin-Buch, E. Germany) Zotter, S.; Sydow, G. *Acta Biol Med Ger* 35(12): 1741-1744; 1976.

Preliminary evidence demonstrating the presence of RNA-dependent DNA polymerase (RDDP) in highly purified intracytoplasmic type-A particles (iAp) from mouse mammary tumors is presented. The iAp were isolated and purified from transplantable CBA/Bln mouse mammary tumors. Assays with <sup>3</sup>H-thymidine-5'-triphosphate showed that purified iAp have both endogenous and exogenous RDDP activities. For optimal endogenous activity, a disruption of the particles and the presence of deoxyribonucleoside triphosphates were required. Preincubation with ribonuclease A and ribonuclease T<sub>1</sub> abolishes or partly abolishes the reaction, suggesting that the enzyme is using the A-particle endogenous RNA as template. The endogenous reaction was linear for up to 6 hr of incubation. The results support the conclusion that iAp from mouse mammary tumors possess RDDP activity that shares its properties with the corresponding enzymes of other RNA tumor viruses. (16 refs.)

77-2667    **Levels of Mammary Tumor Virus in Hormone-dependent and -independent Mouse Mammary Tumor Cells.** (Eng.) Sluyser, M. (Div. Endocrinology, Netherlands Cancer Inst., Antoni von Leeuwenhoek-Huis, Amsterdam, Netherlands) Nouwen, T.; Hilgers, J.; Calafat, J. *Cancer Res* 37(7): 1988-1990; 1977.

Levels of mammary tumor virus particles (types A and B) and virus antigen were assayed in hormone-dependent and -independent mammary tumors of GR mice. Various transplant generations of seven separate tumor lines were investigated. The results indicated that the tumors consisted of different cell clones, each of which exhibited a separate progressive expression and subsequent loss of the mammary tumor virus. When the tumors were transplanted, levels of



B particles first declined in the hormone-dependent cells. In later transplant generations, however, the B particle content of the autonomous cells also dropped. In some tumor lines, this was accompanied by a decrease in viral antigens and/or A particles; in other lines these concentrations remained high. One tumor line (line V), which remained hormone-dependent throughout nine transplantations, was practically devoid of B particles but contained high levels of A particles and mammary tumor antigen. (13 refs.)

- 77-2668 **C-Type and Intracisternal A-Type Virus Particles During Epidermal Carcinogenesis by Tobacco Smoke Condensate in BALB/c Mice.** (Eng.) Bibby, M. C. (Hazleton Labs. Europe Limited, Otley Road, Harrogate, Yorkshire HG3 1PY, England) Smith, G. M. *Br J Cancer* 35(6): 743-751; 1977.

Electron microscopic observations of sequential stages of BALB/c mouse skin carcinogenesis induced by tobacco smoke condensate (SC) and a cyclohexane fraction of tobacco smoke condensate (G) revealed an increase in the incidence of intracisternal A particles within the epidermal cells. Tumors induced by SC (150 mg for 10 wk followed by 200 mg/wk) also contained C-type particles, but these were not seen in tumors induced by G (150 or 200 mg/wk) or after irritant or solvent treatment. There was no evidence of an increase in intracisternal A particles after irritant or solvent treatment. A direct relationship between the proliferation of A particles and the neoplastic growth of BALB/c mouse epidermis appears likely. The data suggest the possible activation of a latent C-type virus by SC. (23 refs.)

- 77-2669 **Mitogen Induction of Murine C-Type Viruses. Effect of Culture Conditions, Age, and Genotype.** (Eng.) Schumann, G. (Res. Dept., Pharmaceuticals Div., CIBA-Geigy Limited, CH-4002 Basel, Switzerland) Moroni, C. *Virology* 79(1): 81-87; 1977.

Various parameters affecting the induction of endogenous C-type virus from mouse spleen cell cultures by lipopolysaccharide (LPS), a B-lymphocyte mitogen, were examined. The relationship between virus induction (assayed by determining reverse transcriptase activity) and mitogenicity (measured by stimulation of intracellular DNA synthesis) was examined in nu/nu spleen cultures treated with 1, 4, 16, or 64 µg/ml LPS. Both phenomena showed a similar dose-response pattern, indicating that virus induction is linked to stimulation of DNA synthesis. 5-Bromo-2'-deoxyuridine (BUdR) enhanced virus induction by LPS in dose-dependent way with an optimum concentration of 5 µg/ml. Cell concentrations between  $2 \times 10^6$  and  $5 \times 10^6$  were optimal for virus release by LPS and LPS/BUdR. Age dependence of virus induction was examined comparing the effects of LPS and BUdR in cultures from

old and young BALB/c mice. Cells from old mice released more virus in response to LPS than those from young mice, but additional BUdR treatment did not enhance virus release. The mitogenic effect of LPS was similar in cultures from old and young mice. Spleen cultures from several mice strains showed differing response patterns. Although BALB/c, C57BL/6, and AKR cultures all released virus in response to LPS, additional BUdR treatment enhanced virus release from BALB/c and C57BL/6 but not from AKR cells. Cultures of 129/J mice were stimulated to divide by LPS but they could not be induced to release virus by this drug alone or in combination with BUdR. These results suggest that in B lymphocytes stimulated by LPS expression of endogenous viral genes is a function of age as well as genotype, unlike their mitogenic response to LPS. (16 refs.)

- 77-2670 **The Spontaneous Release Of Endogenous C-Type Viruses From Myogenic Cells in Culture (Meeting Abstract).** (Eng.) Bendas, C. M. (Hahnemann Medical Coll. and Hosp. Philadelphia, Philadelphia, PA) *Diss Abstr Int [B]* 38(1): 69-70; 1977. (no refs.)

- 77-2671 **The Spontaneous Release of Endogenous Rat Type-C Virus from Myogenic Cells in Culture (Meeting Abstract).** (Eng.) Bendas, C. M. (Dept. Microbiology and Immunology, Hahnemann Medical Coll., Philadelphia, PA 19102) Crowell, R. L.; Goldberg, R. J. *In Vitro* 13(3): 172-173; 1977. (no refs.)

- 77-2672 **Experimental Investigations on the Specificity of Type B and C Oncornaviruses (Meeting Abstract).** (Ger.) Fasske, E. (Borstel, W. Germany) Fetting, R.; Ruhland, D.; Themann, H. *Zentralbl Allg Pathol* 121(3): 298; 1977. (no refs.)

- 77-2673 **Direct Isolation of Xenotropic Retroviruses from the NIH Swiss Mouse Uterus.** (Eng.) Allen, P. T. (NCI, Frederick Cancer Res. Center, NIH, Frederick, MD 21701) Mullins, J. A.; Saviolakis, G. A.; Strickland, J. E.; Fowler, A. K.; Hellman, A. *Virology* 79(1): 239-243; 1977.

Xenotropic C-type retroviruses were isolated from cell-free uterine extracts of normal adult NIH Swiss mice by direct inoculation of mink, cat, and human target cells. Successful isolation occurred in approximately one-half the susceptible target cell cultures (including human rhabdomyosarcoma cells) inoculated with a given extract. These results demon-

strate the presence of fully infectious xenotropic virus particles in the uterus of the normal NIH Swiss mouse. (16 refs.)

77-2674 **Feline Oncornavirus-Associated Cell Membrane Antigen: Expression in Transformed Non-producer Mink Cells** (Eng.) Sliski, A. H. (Dept. Microbiology, Harvard Univ. Sch. Public Health, Boston, MA 02115) Essex, M.; Meyer, C.; Todaro, G. *Science* 196(4296): 1336-1339; 1977.

A nonvirion tumor-specific surface antigen that is induced by a naturally occurring mammalian oncornavirus and is immunologically effective under natural conditions was described for the first time. Mink lung cells transformed by feline sarcoma virus expressed high levels of the feline oncornavirus-associated cell membrane antigen (FOCMA), which is the target for immunosurveillance response in cats against fibrosarcoma and leukemia. Since the transformed cells did not express the major viral structural proteins, it was concluded that FOCMA is independent of the presence of these proteins. Neither murine sarcoma virus nor feline leukemia virus induced FOCMA; thus FOCMA expression was specifically linked with transformation by feline sarcoma virus. (20 refs.)

77-2675 **Ovine Cells: Their Long-Term Cultivation and Susceptibility to Visna Virus**. (Eng.) Torchio, C. (New York State Inst. Basic Res. in Mental Retardation, 1050 Forest Hill Road, Staten Island, NY 10314) Trowbridge, R. S. *In Vitro* 13(4): 252-259; 1977.

Sheep choroid plexus (SCP) cells were subcultured > 120 times, and they underwent > 300 cell generations. These fibroblastic-appearing SCP II-B cells were found to contain ovine-specific antigens, have an absolute plating efficiency of 23%-28%, and be as susceptible to visna virus infection and virus-induced cytopathology as their low-passage-level counterparts. Cultures of low-, relatively high-, and high-passage-level SCP cells produced equivalent amounts of visna virus at similar rates when they were infected with equal amounts of this virus. The passage level of the SCP II-B cells, their elapsed number of cell generations, their possession of ovine-specific antigens, and their full susceptibility to visna virus allow them to be considered an established line of sheep cells. Studies of the mechanisms underlying the in vitro host sheep cell-Lentivirus interaction can now be performed with these SCP II-B cells without being complicated by the relatively short life of ovine cells. The ability to subculture SCP II-B cells for > 120 times suggests that these cells transformed spontaneously and that they possibly contain an oncogene or a provirus. (27 refs.)

77-2676 **Purification and Characterization of the Major Internal Protein (p24) of Bovine Leukemia Virus**. (Eng.) Portetelle, D. (Department de Biologie Molculaire de l'Universite Libre de Bruxelles, Bruxelles, Belgium) Mammerickx, M.; Burny, A.; Cleuter, Y.; Ghysdael, J.; Dekegel, D.; Kettmann, R.; Chantrenne, H. *Arch Int Physiol Biochim* 85(1): 192-193; 1977.

A technique for the isolation of p24 is presented. Using antiserum to this protein, no antibodies against bovine leukemia virus were found in the sera of human cancer patients. (2 refs.)

77-2677 **Properties of Density Gradient-Fractionated Peripheral Blood Leukocytes from Cattle Infected with Bovine Leukemia Virus**. (Eng.) Kenyon, S. J. (Section Viral Oncology, Comparative Leukemia Studies Unit, Sch. Veterinary Medicine, Univ. Pennsylvania, Kennett Square, PA 19348) Piper, C. E. *Infect Immun* 16(3): 893-903; 1977.

Discontinuous bovine serum albumin gradients were used to fractionate peripheral blood WBC from bovine leukemia virus (BLV)-free and BLV-infected cows. The release of infectious BLV and spontaneous incorporation of <sup>3</sup>H-thymidine were not properties of density gradient-fractionated WBC from a BLV-free cow. When WBC from BLV-infected cattle were fractionated, B lymphocytes that spontaneously incorporated <sup>3</sup>H-thymidine could be separated as a distinct subpopulation from B lymphocytes that replicated infectious BLV. Density gradient fractionation of WBC from a cow with lymphosarcoma is also reported. A fall in lymphocyte count at the time of tumor development was attributed to the loss of B lymphocytes that spontaneously incorporate <sup>3</sup>H-thymidine. (10 refs.)

77-2678 **Metrizamide, a New Reagent for Purification of RNA Oncogenic Viruses Associated with Bovine Enzootic Leukemia and Human Leukemias**. (Eng.) Portetelle, D. (Department de Biologie Molculaire de l'Universite Libre de Bruxelles, Bruxelles, Belgium) Ghysdael, J.; Burny, A.; Dekegel, D.; Mammerickx, M.; Prevost, J. M.; Chantrenne, H. *Arch Int Physiol Biochim* 85(1): 194-195; 1977.

The use of Metrizamide gradients for the purification of RNA viruses allowed the recovery of reverse transcriptase and 60 to 70 S RNA. The banding densities of several viruses are presented. (2 refs.)

77-2679 **Induction of Brain Tumors in Hamsters with BK Virus, a Human Papovavirus**. (Eng.) Green-



lee, J. E. (Dept. Neurology, Johns Hopkins Univ. Sch. Medicine, Baltimore, MD 21205) Narayan, O.; Johnson, R. T.; Herndon, R. M. *Lab Invest* 36(6): 636-641; 1977.

The oncogenicity of BK virus for the CNS was studied in newborn Syrian hamsters. The virus was weakly oncogenic after intracerebral inoculation (0.02 ml). Two of 45 hamsters treated with antithymocyte serum developed tumors as opposed to 0/33 untreated hamsters. Both tumors were choroid plexus papillomas, as shown by histologic and electron microscopic examination. Cells cultured from one tumor had the growth characteristics of transformed cells and intranuclear T antigen, but infectious virus could not be rescued. Cultured tumor cells injected sc ( $10^{-6}$  cells) were weakly oncogenic for newborn hamsters; however, tumor induction was enhanced in recipients treated with antithymocyte serum. Tumors did not develop in newborn hamsters inoculated intracerebrally with  $10^5$  tumor cells or in weanling hamsters inoculated sc with large numbers of tumor cells ( $2.5 \times 10^7$  cells). In contrast, tumors developed in all five adults inoculated in the cheek pouch ( $10^7$  cells). The data suggest that the weak oncogenicity of BK virus is due to the immunogenicity of the transformed cells plus attendant rejection by the host. (33 refs.)

**77-2680 Isolation of a Type C RNA Virus from an Established Human Histiocytic Lymphoma Cell Line.**

(Eng.) Kaplan, H. S. (Cancer Biology Res. Lab., Dept. Radiology, Stanford Univ. Medical Center, Stanford, CA 94305) Goodenow, R. W.; Epstein, A. L.; Gartner, S.; Decleve, A.; Rosenthal, P. N. *Proc Natl Acad Sci USA* 74(6): 2564-2568; 1977.

A C-type RNA virus was detected in the culture fluids of a permanently established human histiocytic cell line (SU-DHL-1). In electron micrographs, the virus closely resembled other typical mammalian C-type RNA tumor viruses in size and morphology. Viral RNA-dependent DNA polymerase activity was demonstrated in particles (densities of 1.15 and 1.22 g/ml) in the microsomal cytoplasmic fraction and in pellets of culture fluids. The enzyme is partially inhibited by antibodies to the RNA-dependent DNA polymerases of simian sarcoma virus and RD-114 virus, but not by antibody to the polymerase of murine leukemia virus, suggesting some degree of relatedness to C-type viruses of subhuman primate origin. Typical syncytial microplaques were induced when SU-DHL-1 cells were cocultivated with rat XC cells. Although no focus formation was noted in similarly cocultivated BALB mouse fibroblast UC1-B cell cultures, the numbers of foci induced in rat embryo fibroblasts by murine sarcoma virus were significantly increased by coinfection with the virus from SU-DHL-1 cell culture fluids. No other evidence of infectivity, inducibility, or capacity for helper rescue of defective murine sarcoma virus genomes has been detected to date in cocultivation studies with a spectrum of fibroblastic and other nonlymphoid indicator cell lines of human and other species of origin. (33 refs.)

**77-2681 RNA Tumor Virus-like Activities in Human Prostate: Possible Novel Pharmacologic Approaches.** (Eng.) Arya, S. K. (Dept. Medical Viral Oncology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263) Job, L.; Horoszewicz, J. S.; Zeigel, R. F.; Carter, W. A. *Cancer Treat Rep* 61(2): 113-117; 1977.

The cytoplasmic extracts of human prostatic tissues yielded two classes of particles when centrifuged to equilibrium in a sucrose density gradient, one class banding at a density of 1.15-1.18 g/cm<sup>3</sup> (high-density particles) and another at a density of 1.07-1.14 g/cm<sup>3</sup> (low-density particles). Both bands displayed endogenous DNA polymerase activity that was largely resistant to actinomycin D inhibition. The endogenous DNA products synthesized by the high-density particles gave some indication of high molecular wt RNA:DNA complexes. The tissue extracts from normal, hyperplastic, and neoplastic prostate behaved similarly in these assays. In addition, explant cultures of hyperplastic and neoplastic prostate released or could be induced to release particles by treatment with bromodeoxyuridine. These particles banded at a density of 1.15-1.18 g/cm<sup>3</sup> in a sucrose density gradient and possessed RNA and associated DNA polymerase activity that utilized poly(A):oligo(dT). The results suggest that human prostatic tissues may contain functions analogous to those of known RNA tumor viruses of other species. (7 refs.)

**77-2682 Cytomegalovirus and Cancer of the Prostate: In Vitro Transformation of Human Cells.** (Eng.)

Geder, L. (Dept. Microbiology, Milton S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA 17033) Sanford, E. J.; Rohner, T. J.; Rapp, F. *Cancer Treat Rep* 61(2): 139-146; 1977.

Urogenital tissue specimens were maintained in culture for 2 yr. Epithelioid growth was enhanced with the use of collagenase digestion rather than trypsinization. Twenty of 31 prostate cancer cell cultures survived > 10 in vitro passages, during which time 4/20 demonstrated epithelioid morphology. One epithelioid line (T-157) survived 32 in vitro passages. The cells demonstrated lack of contact inhibition in culture, were slightly positive in acid phosphatase tests, and reacted positively with cytomegalovirus (CMV)-immune sera in indirect immunofluorescence (IF) tests. These cells, which had a normal male karyotype, failed to yield infectious virus and could be reisolated from a nodule induced by the cells when injected sc into weanling athymic nude mice. The serum of the patient from which the tumor cells were derived demonstrated high CMV antibody titers and reacted with the virus-specific membrane and intracellular antigens of CMV-transformed human cells in IF tests. A CMV strain isolated from one of the normal prostate cell cultures established an in vitro long-term persistent infection of human embryo lung cells that resulted in the development of two transformed cell lines. The transformed cells possessed CMV antigenic markers and induced nondifferentiated tumors when transplanted

into athymic nude mice. The results constitute further evidence of the transforming capacity of CMV and suggest that the virus may be oncogenic in its natural (human) host. (2 refs.)

- 77-2683 **Viruses and Renal Carcinoma of *Rana Pipiens*. XV. The Presence of Virus-associated Membrane Antigen(s) on Lucke Tumor Cells.** (Eng.) Naegele, R. F. (Lab. Virology, St. Jude Children's Res. Hosp., P.O. Box 318, Memphis, TN 38101) Granoff, A. *Int J Cancer* 19(3): 414-418; 1977.

The presence of Lucke herpesvirus (LHV)-associated membrane antigens on Lucke tumor cells from four adult frogs, *Rana pipiens*, was examined by immunofluorescence using antiserum prepared against LHV. Intracellular fluorescence, predominantly cytoplasmic, was observed only in fixed smears of virus-containing cells, as observed by electron microscopy. The number of positive cells correlated well with the number of virus-containing cells. Tests for the detection of LHV-specific membrane fluorescence were carried out on in vitro cultured primary tumor cells. Membrane fluorescence was found in 30%-90% of the tumor cells and was not correlated with the presence or absence of virus. A striking reduction in the number of positive-reacting cells was observed with time in primary culture. Absorption of the antiserum with normal frog kidney tissue had no effect on the number of positive cells, but absorption with virus-free tumor reduced the reaction. Absorption with virus-containing tumor eliminated the reaction, suggesting that more antigen is expressed in productively infected cells. It is not known whether every Lucke tumor cell contains a complete genome, but the membrane immunofluorescence tests indicate that a high proportion must carry viral genetic information expressed as virus-specific surface antigen. (19 refs.)

- 77-2684 **Transforming Activity and Antigenicity of an Epstein-Barr-like Virus from Lymphoblastoid Cell Lines of Baboons with Lymphoid Disease.** (Eng.) Rabin, H. (Viral Oncology Program, NCI-Frederick Cancer Res. Center, Post Office Box B, Frederick, MD 21701) Neubauer, R. H.; Hopkins, R. F.; Dzhikidze, E. K.; Shevtsova, Z. V.; Lapin, B. A. *Intervirology* 8(4): 240-249; 1977.

The isolation of an Epstein-Barr virus (EBV)-like herpesvirus from two lymphoid cell lines established directly from baboons with lymphoma is reported. Receptors for activated complement and sheep red blood cells (SRBC) were assayed by a combined rosetting method. An immunofluorescent assay was used to detect Fc receptors. Indirect immunofluorescence was used to test for viral capsid antigen (VCA), early antigen (EA), membrane antigen (MA), and EB nuclear antigen (EBNA). Peripheral blood lymphocytes (PBL) of various species of nonhuman primates and human cord lymphocytes

were used for in vitro transformation tests. The cells of the two baboon lines were positive for complement and FC receptors but they lacked SRBC receptors, indicating a B-cell origin. The cells contained antigens that cross-reacted with EBV, VCA, EA, and MA. The virus was neutralized by anti-MA-positive baboon and human sera. Both lines released virus with in vitro transforming activity for the PBL of several primate species, including humans. Cells of the original lines and transformed cells showed no staining for EBNA. Baboon virus and EBV had different but overlapping in vitro host-cell ranges. The production of EBNA in EBV-transformed cells and its absence in the same cell types transformed by baboon cell virus indicate that EBNA production is a virus-associated function. (14 refs.)

- 77-2685 **Enhanced Oncogenic Behavior of Human and Mouse Cells after Cellular Hybridization with Burkitt Tumor Cells.** (Eng.) Glaser, R. (Dept. Microbiology, Milton S. Hershey Medical Center and Specialized Cancer Res. Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA 17033) Ablashi, D. V.; Nonoyama, M.; Henle, W.; Easton, J. *Proc Natl Acad Sci USA* 74(6): 2574-2578; 1977.

The expression of Epstein-Barr virus (EBV) in somatic hybrids of Burkitt tumor cells and human or mouse cells was studied to determine whether EBV genetic information associated with the capacity to transform the WBC of humans and nonhuman primates could be expressed in nonlymphoblastoid cells. Examination of mouse/Burkitt hybrid cells (CL1D/Raji) suggested that at least one characteristic of cellular transformation (loss of contact inhibition) is expressed only in nonlymphoblastoid cells in which the EBV genome is maintained. In addition, human epithelial Burkitt hybrid cells (D98/HR-1 and D98/Raji) were more oncogenic in athymic mice than the cells of the human epithelial parental line D98 or the Raji Burkitt parent cell line. The HR-1 Burkitt parent cell line was as oncogenic as the hybrid lines, but the time required for induction was much longer. Thus, human epithelial cells show altered growth properties in vitro and in vivo after cellular hybridization with Burkitt tumor cells. (23 refs.)

- 77-2686 **Cell Surface Markers on Epithelial-Burkitt Hybrid Cells Superinfected with Epstein-Barr Virus.** (Eng.) Glaser, R. (Dept. Microbiology, Milton Hershey Medical Center, Pennsylvania State Univ., Hershey, PA 17033) Lenoir, G.; Ferrone, S.; Pellegrino, M. A.; de-The, G. *Cancer Res* 37(7): 2291-2296; 1977.

Attempts were made to superinfect two epithelial-Burkitt hybrid cell lines, D98/HR-1 and D98/Raji, with Epstein-Barr virus (EBV) and to investigate the expression of some cell surface markers, including histocompatibility antigens, and the presence of B-cell markers, such as receptors for the third



complement component and for monkey RBC. Successful superinfection of D98/HR-1 cells with EBV was evidenced by the expression of early antigen and, to a lesser extent, virus capsid antigen. Only a rare D98/Raji cell was positive for early antigen. The histocompatibility antigens of the parental cell lines D98, HR-1, and Raji were expressed on the surfaces of the hybrid cells. Receptors for third complement components b and d were not detected on the hybrid cells or on the D98 or HR-1 cells; they were found, however, on the Raji cells, indicating that EBV receptors and complement receptors can be separated. The significance of the infection of hybrid cells with EBV and the expression of cell surface markers is described. (17 refs.)

- 77-2687 Search for Infectious Epstein-Barr Virus-releasing Cell Lines, with Particular Reference to a New Producer Line, NHAd-60. (Eng.) Sakamoto, K. (Dept. Microbiology, Kumamoto Univ. Medical Sch., Honjo 2-2-1, Kumamoto 860, Japan) *Gann* 68(2): 145-150; 1977.

Thirty-six Epstein-Barr virus (EBV) genome-carrying human lymphoblastoid cell lines, all of which originated from various Japanese individuals, were screened in a search for infectious EBV-producer cell lines. Viral infectivity was detected by two methods: assay for human WBC-transforming activity and assay for early antigen (EA)-forming activity. Three cell lines from different sources released WBC-transforming activity in the culture fluid, but no cell line yielded EA-inducing activity. One of the three cell lines, NHAd-60 (from adenoid tissue), released a relatively large amount of infectious virus ( $10^2 - 10^3$  of 50% of the transforming dose of cord WBC). The NHAd-60 cell line also possessed a high frequency of cells with EBV-associated antigens, 5% viral capsid antigen and 20% membrane antigen-positive cells. The cells did not have EBV receptors. NHAd-60 virus-transformed cord cell lines contained a higher frequency of IgA-producing cells compared to transformed cell lines from the same WBC samples infected with two other viral strains. It is not clear whether this characteristic reflects the tissue of origin of the virus, because NHAd-60 had originated from IgA-rich adenoid tissue. (22 refs.)

- 77-2688 Isolation of a Herpes Virus Specific DNA Polymerase from Tissues of American Burkitt's Lymphoma (Meeting Abstract). (Eng.) Allauden, H. S. (Dept. Pharmacology and Medicine, Yale Univ., New Haven, CT 06510) Bertino, J. R. *Proc Am Assoc Cancer Res* 18: 62; 1977. (no refs.)

- 77-2689 Unusual Prevalence of Antibodies to Epstein-Barr Virus Early Antigen in Ataxia Telangiectasia

(Letter to Editor). (Eng.) Joncas, J. (Dept. Virology, Institut Armand-Frappier, Montreal, Province of Quebec, Canada) Lapointe, N.; Gervais, F.; Leyritz, F.; Wills, A. *Lancet* 1(8022): 1160; 1977.

Of 16 patients with ataxia telangiectasia, 8 had antibodies (titers of 1/5 to 1/80) to Epstein-Barr (EBV) early antigen (EA), compared with 0/16 matched controls. The prevalence of EBV-EA antibodies in the families of the EA-positive patients was 19% (8/42 unaffected members). The possible significance of persisting EA antibodies for tumor development in EBV infection is discussed. (8 refs.)

- 77-2690 A Study on the Relation Between the Epstein-Barr Virus and Some Forms of Malignant Tumors in Children. (Eng.) Gourtsevich, V. E. (Oncological Res. Center, Acad. Medical Sciences USSR, Moscow, 115 478, USSR) Stepina, V. N.; Mazurenko, N. P.; Durnov, L. A.; Yarymova, N. M.; Yermakov, Ya. S. *Neoplasma* 23(5): 533-539; 1977.

Sera from children with different neoplasms were studied by indirect immunofluorescence for the presence of antibodies to the viral capsid antigen of Epstein-Barr virus (EBV). Serum samples were obtained from 48 children with Wilms' tumor, teratoblastoma, reticulosarcoma, neuroblastoma, or sarcoma and from 21 children with benign tumors. P3HR-1 cells, a Burkitt's lymphoma clone, were used as test cells for viral capsid antigen synthesis. No significant increase (in comparison with controls) in EBV antibodies was demonstrated in any of the tumor groups. The spread of EBV infection in the different patient groups and controls ranged between 83% and 100%. (25 refs.)

- 77-2691 Spontaneous and Induced Patterns of the Epstein-Barr Virus (EBV) Cycle in Human Lymphoma Cell Lines and Their Somatic Cell Hybrids (Meeting Abstract). (Eng.) Clements, G. B. (Dept. Pathology, Univ. Glasgow, Western Infirmary, Glasgow G11 6NT, Scotland) Klein, G. *Br J Cancer* 35(2): 245-246; 1977. (no refs.)

- 77-2692 Establishment and Characterization of a Human Epstein-Barr Virus (EBV) Negative Lymphoblastoid Cell Line Bearing Leukemia Associated Antigen(s) (Meeting Abstract). (Eng.) Rosenfeld, C. (Institut de Cancerologie et d'Immunogenetique, INSERM, 94800, Villejuif, France) Venuat, A. M.; Choquet, C.; Goutner, A.; Kayibanda, B.; Pico, J. L.; Dore, J. F.; Greaves, M. *In Vitro* 13(3): 172; 1977. (no refs.)

77-2693 Selective Inhibition of the Outgrowth of Epstein-Barr Virus-carrying Cell Lines from Leucocytes of Infectious Mononucleosis Patients (Meeting Abstract). (Eng.) Rickinson, A. B. (Dept. Pathology, Medical Sch., Univ. Bristol BS8 1TD, England, Bristol) Epstein, M. A. *Br J Cancer* 35(2): 248; 1977. (no refs.)

77-2694 Does Influenza and Contact with Malignant Neoplasia Predispose to Leukaemia? (Eng.) Kemmoona, I. (Dept. Medicine, Limerick Regional Hosp., Dooradoyle, Limerick, Ireland) *Ir J Med Sci* 146(5): 132-135; 1977.

Five patients developed leukemia or lymphoma after being exposed while suffering from severe influenza infection to cancer patients (four relatives, one neighbor). The interval of 2 to 8 wk between onset of influenza and development of leukemia could represent an incubation period. The influenza may have caused increased susceptibility to a transmissible leukemic agent by depressing immune and leukocyte functions. (23 refs.)

77-2695 Fragmentation Studies Revealing Repetitious Terminal Sequences in Herpes Simplex Virus DNA as Shown by Electron Microscopy and Ultracentrifugation. (Eng.) Muller, U. (Institut für Virusforschung am Deutschen Krebsforschungszentrum, Postfach 101949, D 6900 Heidelberg, W. Germany) Zentgraf, H.; Kaerner, H. C. *Cytobiologie* 14(1): 148-164; 1976.

The structure of the termini of a large herpes simplex virus (HSV) DNA molecule was studied by electron microscopy and ultracentrifugation. DNA molecules extracted from HSV type 1 ANG (HSV ANG) had a molecular wt of  $101 \times 10^6$  daltons, as determined by sedimentation on sucrose gradients. When HSV ANG DNA that had been stored at low concentrations ( $< 1 \mu\text{g DNA/ml}$ ) and in low salt buffer was passed through the sucrose gradients, two fractions with molecular wts of  $97 \times 10^6$  and  $4.6 \times 10^6$  daltons were observed. This suggests that spontaneous fragmentation of intact HSV ANG DNA occurs near one or both ends of the molecule. Self-annealing of these terminal fragments led to the formation of large linear and branched aggregates measuring up to  $70 \times 10^6$  daltons. Electron microscopy results indicate that the terminal fragments contain repeats of short single-stranded (ss) nucleotide sequences (between 12 and 80 bases) interspersed at regular intervals of about 240 base pairs. It is estimated that the terminal repetitious segments of HSV ANG DNA account for a minimum of 2,400 base pairs, or 1.5% of the viral genome. According to a tentative model of the sequence of the HSV ANG DNA termini, both inverted and ordinary repetitions would explain the formation of rings as well as of linear and branched annealing products. (34 refs.)

77-2696 Studies of the DNA Excision Repair in Lymphocytes of Patients with Recurrent Herpes Simplex (Meeting Abstract). (Eng.) Fanta, D. (II. Dept. Dermatology, Univ. Vienna, Res. Center Seibersdorf-Inst. Biology, "L. Boltzmann Institut zur Erforschung infektiöser venerodermatologischer Erkrankungen", Vienna, Austria) Tobaloglou, A.; Altmann, H.; Soltz-Szots, J. *Arch Dermatol Res* 258(1): 109; 1977. (no refs.)

77-2697 Herpes Simplex Virus Tumor-associated Antigens in Cancer Patients. (Eng.) Tarro, G. (Div. Virology, Cattedra di Virologia Oncologica, la Facolta di Medicina e Chirurgia, Ospedale "D. Cotugno," Naples, Italy) Di Gioia, M.; Cocchiara, R.; Smeraglia, R.; Giordano, G. G.; Tripodi, A. *Tumori* 62(6): 615-622; 1976.

Studies of herpes simplex virus (HSV) types 1 and 2 antigens in tissue culture and selected cancer cells are reported, as are studies of specific antibodies to nonvirion (NV) antigens in immunized guinea pigs and in sera derived from certain cancer patients. HSV-1 and HSV-2 NV antigens consisted of more than one component for which immunized guinea pigs produced distinct antibodies. HSV-induced markers from cells undergoing lytic infection by the virus and from viable cells from squamous cell carcinoma of the head and neck and the urogenital tract could be separated by polyacrylamide gel electrophoresis (PAGE). The percentage of sera from patients with head and neck cancers that reacted with PAGE cancer region 3 from leukoplakias and lip carcinomas increased according to the sequential pathologic changes of the neoplastic tissue tested. The specificity of the antibody to the antigen from the cancer cells was less high than that of the antibody to the antigen from HSV-infected cells. The use of these PAGE-separated antigens would eliminate the need for removal of the virion antibody from cancer sera prior to testing for NV-specific antibody. The finding of HSV NV antigens in selected tumors represents a step forward in the implication of these viruses in human cancer. (15 refs.)

77-2698 Herpes Simplex Virus DNA Sequences in Transformed Cells (Meeting Abstract). (Eng.) Frenkel, N. (Dept. Biology, Univ. Chicago, 103 E. 57th St., Chicago, IL 60637) *Hoppe Seylers Z Physiol Chem* 358(4): 421; 1977. (no refs.)

77-2699 The Combined Effect of Herpes Simplex Virus Type 2 and a Polychlorinated Biphenyl (Arochlor 1242) on Human Chromosomes: Induction of Sister



Chromatid Exchanges (Meeting Abstract). (Eng.) Nicholas, A. H. (Oregon State Univ., Corvallis, OR 97331) *Diss Abstr Int [B]* 38(1): 74-75; 1977. (no refs.)

77-2700 Structure and Nucleotide Sequence Studies of Adeno-associated Virus DNA (Meeting Abstract). (Eng.) Fife, K. H. (Johns Hopkins Univ., Baltimore, MD 21218) *Diss Abstr Int [B]* 38(1): 70-71; 1977. (no refs.)

77-2701 Adenovirus--Integration and Oncogenicity (Meeting Abstract). (Eng.) McDougall, J. K. (Cold Spring Harbor Lab., P.O. Box 100, Cold Spring Harbor, NY 11724) *Hoppe Seylers Z Physiol Chem* 358(4): 420-421; 1977. (5 refs.)

77-2702 Mechanisms of Genetic Exchange Between SV 40 and Adenoviruses (Meeting Abstract). (Eng.) Sambrook, J. (Cold Spring Harbor Lab., P.O. Box 100, Cold Spring Harbor, NY 11724) *Hoppe Seylers Z Physiol Chem* 358(4): 420; 1977. (no refs.)

77-2703 Cross-Reaction Between Antigens of Human Myelogenous Leukemia and Mason-Pfizer Monkey Virus. (Eng.) Kim, B. S. (Dept. Microbiology-Immunology, Northwestern Univ. Medical Center, Chicago, IL 60611) *Eur J Cancer* 13(7): 721-728; 1977.

An antigen similar to a major protein (p25) of Mason-Pfizer monkey virus (MPMV) was found in the breast cancers of 2/9 women and in the splenic tissue or the peripheral WBC from 12/12 patients with myelogenous leukemias. The antigen was not detected in other tumors or normal tissues. The cross-reactivity was detected by inhibition of the indirect quantitative radioimmunoprecipitation of <sup>125</sup>I-labeled purified MPMV p25 and anti-MPMV antiserum. Particles banding in a density region of 1.15-1.17 g/ml, the density of the known animal leukemia viruses, were used as precipitation inhibitors. An antigen isolated from the spleen of a patient with myelogenous leukemia using an anti-p25 immunoabsorbent column was similar in net electrical charge and molecular wt to MPMV p25. These results suggest that a viruslike particle bearing some antigenic relatedness to MPMV may be potentially important in the pathogenesis of human myelogenous leukemia. (23 refs.)

77-2704 Characterization of Single Cell Clones Derived from Mason Pfizer Virus-Transformed Rhesus Monkey Cell Cultures (Meeting Abstract). (Eng.) Brown, B. L. (NCI Frederick Cancer Res. Center, P.O. Box B, Frederick, MD 21701) Fine, D. L.; Kmetz, J. P. *In Vitro* 13(3): 175; 1977. (no refs.)

77-2705 Morphological Phenotype of Temperature-sensitive Mutants of Simian Virus 40 in Productive Infection. (Eng.) Kimura, G. (Inst. Cancer Res., Univ. Kyushu Sch. Medicine, Maidashi, Fukuoka 812, Japan) Katsumoto, T.; Itagaki, A. *Virology* 79(2): 355-368; 1977.

Temperature-sensitive (ts) mutants of simian virus 40 (SV40) from the complementation Groups I (viral-DNA positive and V-antigen positive), II (viral-DNA positive and V-antigen negative), and III (viral-DNA negative and V-antigen negative) were examined by electron microscopy for their ability to synthesize physical virus particles at the nonpermissive temperature (40 C) in productive infection. Examinations of thin sections of infected cells revealed that the Group I mutants were not ts for the synthesis of physical virus particles in terms of the proportion of cells synthesizing virions, approx number and mode of arrangement of virions in the virus-producing cells, and morphology of virions present in the cells. Mutants of complementation Groups II and III were ts for the synthesis of virions or virion-related structures. The virus particles of the Group I mutants produced at 40 C, in contrast to those produced at the permissive temperature (33 C), were readily destroyed after the cells were lysed by repeated freezing and thawing or by sonication; however, the lysate still retained V antigen in an amount similar to that in lysates from the mutant-infected cells at 33 C and from wild-type-infected cells at 33 and 40 C. Temperature-shift experiments showed that the ts event in Group I mutant infection occurs about the time of the appearance of new infectious virions, suggesting that the mutation affects the final stage of virus maturation. The function of the Group II mutant is affected about 10 hr prior to the time of the appearance of new infectious virions at 33 C, although the mutant can be regarded as a late mutant because of the heat lability of the virions produced at 33 C and because viral DNA synthesis is normal at 40 C. The Group III mutant temperature suggests that the three genetically distinct mutant groups represent three functionally distinct processes in the replicative cycle of SV40. (27 refs.)

77-2706 Structural Studies on the SV40 DNA Genome. (Eng.) Contreras, R. (Laboratorium voor Moleculaire Biologie, Rijksuniversiteit Gent, Ghent, Belgium) Rogiers, R.; Thys, F.; Van de Voorde, A.; Van Heuverswyn, H.; Van Herreweghe, J.; Volckaert, G.; Ysebaert, M.; Fiers, W. *Arch Int Physiol Biochim* 85(1): 158-160; 1977.

A detailed structural map of the SV40 genome is presented based on the work of several investigators. The cleavage sites of 10 or more additional restriction enzymes were located, and the small fragments thus provided were investigated by nucleotide sequence analysis. (2 refs.)

**77-2707 The Structure of SV40 DNA and SV40 DNA Recombinant Molecules (Meeting Abstract).** (Eng.) Nathans, D. (Dept. Microbiology, Johns Hopkins Univ. Sch. Medicine, Baltimore, MD 21205). *Hoppe Seylers Z Physiol Chem* 358(4): 419-420; 1977. (4 refs.)

**77-2708 Characterization of a Soluble Simian-Virus-40 Transcription Complex.** (Eng.) Gariglio, P. (Institut de Chimie Biologique, Faculte de Medecine de l'Universite Louis-Pasteur, 11 Rue Humann, F-67085 Strasbourg-Cedex, France) Mousset, S. *Eur J Biochem* 76(2): 583-590; 1977.

A nucleoprotein complex able to achieve viral transcription was isolated from the nuclei of monkey cells infected with simian virus 40 (SV40). This complex contains SV40 DNA and RNA polymerase II molecules, which initiated the transcription during viral development. Molecular hybridization experiments demonstrated that most of the templates active in SV40 transcription can be dissociated from the host DNA. In conditions in which supercoiled SV40 DNA form I sediments at 21S, the transcription complex has a sedimentation coefficient of about 25S. Inhibition of viral DNA synthesis by cytosine arabinonucleoside or chloroquine does not affect the activity of the transcription complex, which suggests that replicating molecules are not required for viral RNA synthesis and that SV40 DNA form I could serve as template for late SV40 transcription. A large fraction of the RNA synthesized in vitro remains associated with the SV40 DNA template in a cesium sulfate density gradient. The RNA chains

produced by the complex are heterogeneous in size, and most are as large as or larger than the viral genome. (34 refs.)

**77-2709 Cyclic AMP-Binding Proteins in Normal and Virus-Transformed Fibroblasts.** (Eng.) Wigglesworth, N. M. (Imperial Cancer Res. Fund Lab., Lincoln's Inn Fields, London WC2, United Kingdom) Mastro, A.; Bourne, H. R.; Rozengurt, E. *Arch Biochem Biophys* 180(2): 258-263; 1977.

The hypothesis that altered growth regulation in SV40-transformed fibroblasts may be due to a biochemical lesion affecting cytosol receptor for cyclic AMP was tested. Normal cultured fibroblasts used after two passages and transformed cells from mouse and hamster possessed equivalent amounts of total cyclic AMP receptor. Apparent affinities for cyclic AMP and distribution among isoenzymic forms of cyclic AMP-dependent protein kinase were similar in transformed and normal fibroblasts. Untransformed 3T3 cells displayed only a single peak of cyclic AMP-binding activity, as resolved by ion-exchange chromatography. Thus, these findings do not support the hypothesis that defective binding of cyclic AMP is essential for loss of growth control in vitro. (36 refs.)

\* (Rev): 77-2432, 77-2433, 77-2434, 77-2435, 77-2436, 77-2437, 77-2438.

\* (Chem): 77-2557, 77-2564, 77-2578.

\* (Immun): 77-2712, 77-2713, 77-2721, 77-2722, 77-2723, 77-2724, 77-2726, 77-2727, 77-2728, 77-2729, 77-2730, 77-2731, 77-2732, 77-2733, 77-2734, 77-2739, 77-2754, 77-2761, 77-2762, 77-2763, 77-2765, 77-2774, 77-2775, 77-2776, 77-2780, 77-2782, 77-2784, 77-2786, 77-2787, 77-2792, 77-2793, 77-2794, 77-2796, 77-2803, 77-2804, 77-2805, 77-2818.

\* (Path): 77-2856, 77-2857.

\* (Epid): 77-2894.



- 77-2710 **Immunological Studies of Patients with Asbestosis. II. Studies of Circulating Lymphoid Cell Numbers and Humoral Immunity.** (Eng.) Kagan, E. (American Red Cross Blood Program, Natl. Headquarters, Washington, DC 20006) Solomon, A.; Cochrane, J. C.; Kuba, P.; Rocks, P. H.; Webster, *Clin Exp Immunol* 28: 268-275; 1977.

As part of an overall assessment of immunological function, several aspects of humoral immunity and circulating lymphocyte subpopulations were evaluated in 26 patients with radiographic evidence of parenchymal asbestosis. Statistical comparisons were made between the patient group and a comparable group of 45 controls. Both the percentages and absolute numbers of circulating T lymphocytes were significantly reduced in the patient group compared with controls. Significant elevations of salivary secretory IgA and of serum IgA, IgG, IgM, and IgE were noted among the patients compared with controls. Non-organ-specific autoantibodies and cold-reactive lymphocytotoxins were present in high frequency in the sera of the patients. Neoplasms were detected in four of the patients. The possible significance of these findings is discussed. (27 refs.)

- 77-2711 **The Adjuvant Active Fraction of Delipidated Mycobacteria.** (Eng.) Hiu, I. J. (Institut de cancérologie et d'immunogenetique, CNRS, Hopital Paul-Brousse, 14 Avenue Paul-Brousse, 14 Avenue Paul-vaillant-couturier, 94-Villejuif, France) *Nature* 267(5613): 708-709; 1977.

Data are presented that suggest that the stimulation of cell-mediated and humoral responses by Freund's complete adjuvant is due to the lipoglycopeptide fraction (LG) extracted from mycobacterial delipidated cells. In BD F<sub>1</sub> (DBA/2 x C57BL/6) mice, LG isolated from *Mycobacterium tuberculosis* induced strong delayed skin reactions to ovalbumin (1 mg) and increased the number of plaque-forming spleen cells after immunization with sheep RBC (10<sup>9</sup> ip). The ability of LG to act as an adjuvant of both cell-mediated and humoral responses was related to the balance between its glycopeptide and lipid moieties, since (1) a decrease in the hydrosolubility of the glycopeptide moiety by acylation of free hydroxyl groups destroyed the cellular activity of LG and (2) an increase in the liposolubility of the lipid moiety by catalytic hydrogenation inhibited the humoral immune response. A derivative in which both the lipid and glycopeptide moieties were modified was completely inactive for both immune responses. (10 refs.)

- 77-2712 **Immunogenicity and MuMTV-like Antigenicity of Human Breast Cancer Tissues.** (Eng.) Black, M. M. (New York Medical Coll., Flower & Fifth Ave. Hosps., New York, NY 10029) *Contemp Top Immunobiol* 6: 239-262; 1977.

A review of studies on the immunogenicity of breast cancer resulted in the following conclusions: (1) patients vary with regard to the biologically significant immunogenicity of their cancer tissue and the specific hypersensitivity responses to their cancer tissue; (2) prognostically significant immunogenicity and hypersensitivity are maximally present during the preinvasive stage of the disease; and (3) disease progression is associated with or reflects a loss of immunogenicity and/or specific hypersensitivity. In WBC migration tests, positive responses were obtained most regularly when the target was autologous in situ cancer tissue from Stages 0 and I cases. These responses were least common when the target was invasive breast cancer tissue from Stage II breast cancer patients. The response of WBC to autologous invasive breast cancer tissues having similar nuclear grades correlated with the lymphoreticuloendothelial responses. Polyacrylamide gel electrophoresis of the protein components of breast cancer tissue eluates suggests that the antigenic properties of these tissues are correlated with protein components that have antigenic and physicochemical similarities to murine mammary tumor virus (MuMTV) proteins. It appears from the available data that MuMTV-like antigenicity and protein patterns may provide an approach toward the biologically significant characterization of individual cancers. (58 refs.)

- 77-2713 **Molecular Studies of Immunopathology in Plasmacytoma. Possible Role of Intracisternal A Particles.** (Eng.) Giacomoni, D.; Katzmman, J. In: *Immune RNA in Neoplasia*. Fink, M. A., ed. (New York: Academic Press, Inc.) pp. 85-94; 1976.

Immunodeficiency and cell conversion were induced by injecting mice with RNA-rich extracts (200-500 µg/mouse) obtained from the plasma of plasmacytoma-mice or from the tumor cells themselves. Cell-fractionation studies were conducted to determine the humoral factors responsible for these phenomena. Plasma from plasmacytoma-mice showed both cell-converting and immunosuppressive activities that could be found in the interface between the 20% and 40% sucrose layers (20/40 fraction). Plasmacytoma subcellular fractions enriched in intracisternal A particles also contained both activities. In an in vitro assay, 20-40 µg of poly A-containing RNA were sufficient to change the surface immunoglobulin

of a significant portion of normal mouse spleen cells. The sedimentation velocity of the RNA molecules with both activities was in the 12S-18S and 40S-50S regions. Although the RNA moiety of the A particles seems to be responsible for the activities, the possibility of two different factors cannot be ruled out. (19 refs.)

77-2714    **Studies on the Pathogenesis of an Immune Defect in Multiple Myeloma.** (Eng.) Paglieroni, T. (Dept. Internal Medicine, Section Hematology and Oncology, Univ. California at Davis Sch. Medicine, Davis, CA 95616) MacKenzie, M. R. *J Clin Invest* 59(6): 1120-1133; 1977.

Peripheral blood lymphocytes from 32 patients with multiple myeloma (9 untreated), 15 patients with benign monoclonal gammopathy (BMG), and 30 healthy subjects were tested for their antigen-binding capacity, in vitro proliferative response to antigens and mitogens, and mitogen- or antigen-stimulated immunoglobulin (Ig) production. The effects of coculture of myeloma lymphocytes and normal lymphocytes on Ig production and mixed WBC reactions were also explored. All myeloma patients had normal numbers (3-8/5,000 cells) of cells capable of binding radiolabeled pneumococcal polysaccharide, tetanus toxoid, or diphtheria toxin. However, the proliferative response to these antigens as well as to pokeweed mitogen and streptokinase-streptodornase was depressed, as measured by <sup>125</sup>I-5-iodo-2'-deoxyuridine uptake. Ig production in response to a specific antigen in myeloma lymphocytes was 30%-80% less than in normal lymphocytes. Ig synthesis and mixed WBC responses by normal lymphocytes could be suppressed by myeloma lymphocytes. Multiple suppressor populations were present. Thus, the immune defect in myeloma is beyond the antigen recognition step and involves both the proliferation of antigen-sensitive cells and Ig production. Further suppressive effects are imposed on normal cells, implying defects in immunoregulation in this disease. Patients with BMG did not show many of the defects seen in myeloma. (33 refs.)

77-2715    **Cell Lines from Angio-immunoblastic Lymphadenopathies (AIL) and Immunoblastic Sarcomas (IS) (Meeting Abstract).** (Eng.) Yerganian, G. (Sidney Farber Cancer Inst., Boston, MA 02115) Paika, I. J.; Penta, A.; Liu, T.; Nell, M. A. *In Vitro* 13(3): 175; 1977. (no refs.)

77-2716    **Immune Function in the Leukemia Prone Mouse Strain AKR (Meeting Abstract).** (Eng.) Panfili, P. R. (Purdue Univ., Lafayette, IN 47907) *Diss Abstr Int [B]* 37(8): 3865; 1977. (no refs.)

77-2717    **The Immunologic Activity of Subcellular Fractions and Soluble Protein from a Murine Fibrosarcoma (Meeting Abstract).** (Eng.) Miller, L. S. (Ohio State Univ., Columbus, OH 43210) *Diss Abstr Int [B]* 37(8): 3864; 1977. (no refs.)

77-2718    **Massive Lymphoproliferation and Autoimmunity Controlled by Single Genes (Meeting Abstract).** (Eng.) Murphy, E. D. (The Jackson Lab., Bar Harbor, ME 04609) Roths, J. B.; Lane, P. W. *Proc Am Assoc Cancer Res* 18: 157; 1977. (no refs.)

77-2719    **Immune Mechanisms in Human Colon Cancer (Meeting Abstract).** (Eng.) Hahn, W. V. (Dept. Medicine, Univ. California, La Jolla, CA 92093) Kagnoff, M. F.; Lewis, S.; Trefts, P. *Gastroenterology* 72(5/Part 2): 1067; 1977. (no refs.)

77-2720    **Studies on the Prostate and Testis as Immunologically Privileged Sites.** (Eng.) Whitmore, W. F. (Harvard Program in Urology, Harvard Medical Sch., Peter Bent Brigham Hosp., 721 Huntington Ave., Boston, MA 02115) Gittes, R. F. *Cancer Treat Rep* 61(2): 217-222; 1977.

The immunology of the prostate and testis was studied by observing allograft survival in rats and by measuring humoral antibody and delayed hypersensitivity responses (DHR) to tissue antigens in rabbits. The strain combinations of Buffalo (Bu)→Lewis (Le) and Le→Fisher (Fi) rats were used for the allograft studies. The DHR to tissue antigen(s) was diminished in organs without demonstrable afferent lymphatics (ie, prostate, anterior eye chamber), but the humoral response was unaffected. Rats showed prolonged allograft survival in the testis and anterior eye chamber, with intermediate survival in the prostate and muscle, and abbreviated survival of orthotopic grafts. The wide spectrum of allograft survival was more evident in the Le→Fi combination than in the more immunogenic Bu→Le combination. The extraordinary incidence of prostatic carcinoma with increasing age is discussed in light of these data. Latent carcinoma may arise because of an oversight of immune surveillance and may be kept "latent" by a humoral response. The apparent paradox of the testis as an immunologically privileged site remains unexplained. (21 refs.)

77-2721    **Studies on Specific Humoral Immunity in Leukemia.** (Eng.) Bergolz, V. M. (P. A. Herzen Oncological Res. Inst., Moscow, USSR) *Neoplasma* 23(5): 457-462; 1977.

Studies of humoral immunity in 47 patients with acute leukemia and in leukemic BALB/c, C57Bl/6, and CC57BR/



MV mice are summarized. The animal studies established that the immunologic mechanism of viral leukemogenesis involves interrelations between cytotoxic and blocking antibodies. The existence of a specific humoral immune response (immunoglobulins with the properties of antibodies) was demonstrated in 42.6% of the leukemia patients. Different forms of specific humoral immunity manifested in acute myeloid leukemia and acute lymphoid leukemia were distinguished (nonreactive, nonspecific, cytotoxic, blocking, and mixed forms). (7 refs.)

**77-2722 The Influence of Humoral and Cellular Immune System on Chickens with Marek's Disease (Meeting Abstract).** (Eng.) Shieh, H. K. (Univ. Massachusetts, Amherst, MA 01002) *Diss Abstr Int [B]* 38(1): 94-95; 1977. (no refs.)

**77-2723 Immune Response to Rauscher Virus-induced Leukemia in DBA Mice. I. Role of Cellular and Humoral Immunity in Spontaneous Regression.** (Eng.) Toth, F. D. (Inst. Microbiology, Univ. Medical Sch., 4012 Debrecen, Hungary) Gomba, S.; Vaczi, L.; Kasa, M.; Jako, J. *Neoplasma* 23(5): 471-481; 1977.

DBA/1 and DBA/2 mice infected with Rauscher leukemia virus (0.2 ml ip) developed a biphasic erythroleukemia. Transitory regression of the disease was closely associated with the appearance of tumor-specific antibodies on day 10 postinfection, and exacerbation was preceded by a gradual decrease in antibody titer after day 45. The antibody-dependent cellular cytotoxicity appeared earlier than the complement-dependent cytotoxicity (day 18). Moreover, antibody titers responsible for antibody-dependent cytotoxicity were higher than those of antibodies mediating complement-dependent cytotoxicity. The results suggest that antibody-dependent cytotoxicity is mainly responsible for tumor cell rejection. (22 refs.)

**77-2724 Mechanisms of Protection or Enhancement Produced by Pyran Against Friend Leukemia Virus (Meeting Abstract).** (Eng.) Schuller, G. B. (Virginia Commonwealth Univ./Medical Coll. Virginia, Richmond, VA) *Diss Abstr Int [B]* 37(8): 3784-3785; 1977. (no refs.)

**77-2725 Immunization Against Gross Leukemia in Inbred Mice (Meeting Abstract).** (Eng.) Mariani, T. (Dept. Lab. Medicine and Pathology, Univ. Minnesota, Minneapolis, MN 55455) Landucci, G. *Proc Am Assoc Cancer Res* 18: 63; 1977. (no refs.)

**77-2726 The Neutralization of Polyoma Virus (Meeting Abstract).** (Eng.) O'Hara, M. K. (Univ. Iowa City, IA 52240) *Diss Abstr Int [B]* 37(12/Part 1): 598-1977. (no refs.)

**77-2727 Isolation of Virus-free *Herpesvirus saimiri* Antigen-positive Plasma Membrane Vesicles.** (Eng.) Pearson, G. R. (Dept. Microbiology, Mayo Clinic Foundation, Rochester, MN 55901) Scott, R. E. *Proc Nat Acad Sci USA* 74(6): 2546-2550; 1977.

Experiments were undertaken to determine whether virus free, antigen-positive plasma membrane vesicles could be isolated from owl monkey kidney cells infected with *Herpesvirus saimiri* ( $10^5$  plaque-forming units/ml). The results demonstrate that vesicles can be produced by using a vesiculation fluid containing 25 mM formaldehyde/2 mM dithiothreitol. Electron microscopy revealed that these vesicles were free of detectable virus particles. Vesicles prepared from the infected cells contained virus-induced membrane antigens, as shown by membrane immunofluorescence and by inhibition of antibody-dependent lymphocyte cytotoxicity. Nonhuman primates (3 marmosets and 1 baboon) immunized with vesicles produced antibodies to these membrane antigens, late cytoplasmic antigens, and neutralizing antibodies. Infectious virus was not demonstrated in these vesicles by cocultivation with owl monkey kidney cells or by the inoculation of cotton-top marmosets. Furthermore, no DNA could be demonstrated in vesicles prepared from *Herpesvirus*-infected owl monkey kidney cells. The implication of these findings in relation to the question of a virus-free membrane vaccine against *Herpesvirus* infections is discussed. (38 refs.)

**77-2728 Development of Cellular Anti-tumor Immunity in Chickens Bearing Tumors Induced by Rous Sarcoma Virus.** (Eng.) Israel, E. (Lady Davis Inst. for Medical Res., Jewish General Hosp., Montreal, Quebec, Canada) Wainberg, M. A. *J Immunol* 118(6): 2237-2242; 1977.

Peripheral lymphocyte stimulation in response to antigen was used to demonstrate the existence of cell-mediated antitumor immunity in chickens bearing tumors induced by the B77 (subgroup C), Prague (subgroups A and B), and Schmidt-Ruppin (subgroups A and D) strains of Rous sarcoma virus. The expression of this antitumor response against virus-containing transformed cell culture supernatant fluids and cell extracts varied among animals in terms of time after virus inoculation. Animals whose tumors had completely regressed rapidly lost the ability to mount this continuing immunity. Supernatant fluids and extracts of virus-transformed cells were occasionally stimulatory to lymphocytes of normal chickens, and lymphocytes of tumor-bearing animals were sometimes responsive to the supernatant fluids of normal cells. Thus, normal chickens may have endogenous levels of cell-mediated immunity against viral and/or virus-associated tumor antigens. (34 refs.)

77-2729 **Immunostimulation of Tumor Induction by Moloney Sarcoma Virus (MSV) (Meeting Abstract).** (Eng.) Murasko, D. M. (the Jackson Lab., Bar Harbor, ME 04609) Prehn, R. T. *Proc Am Assoc Cancer Res* 18: 1977. (no refs.)

77-2730 **Modifying Effects of a Benign Virus on the Malignant Process and the Role of Physiological Stress on Tumor Incidence.** (Eng.) Riley, V.; Spackman, J. In: *Modulation of Host Immune Resistance in the Prevention or Treatment of Induced Neoplasias*. NCI and John E. Garatty International Center for Advanced Study in the Health Sciences. (Bethesda, MD) pp. 319-336; 1977.

The ip administration of lactate dehydrogenase (LDH) virus, which is a benign passenger in most classical transplantable mouse tumors and oncogenic virus preparations, elevated corticosterone levels significantly in three strains of 6- to 10-month-old female mice. These elevations apparently caused virus involution and lymphocytopenia. When LDH virus was administered together with oncogenic viruses, tumor growth was significantly enhanced, presumably because of a decrease in cell-mediated immunity. In genetically susceptible C3H/HeJ female mice carrying the Bittner mammary tumor virus from birth, tumor incidence was dramatically reduced from about 90% in mice housed under sustained stress to < 10% in mice housed in plastic cages held in specially designed protective, enclosed, ventilated shelves that minimize stress induction. The data suggest that social and physiological stress may compromise immunological competence and thus enhance malignancy. Reexamination of earlier experimental data on tumor induction in mice is recommended. (102 refs.)

77-2731 **Immunogenetic Studies on Meth-A-Vaccinia Tumour Cells In Vivo and In Vitro.** (Eng.) Garrido, F. (Tissue Immunology Unit, London Hosp. Medical School, Turner St., London E1 2AD, England) Schmidt, W.; Festenstein, H. *J Immunogenet* 4(2): 115-125; 1977.

The expression of H-2 antigenic specificities on Meth-A-vaccinia tumor cells and Meth A cells was compared using an assay based on inhibition of the complement-dependent lymphocytotoxicity of <sup>51</sup>Cr-labeled normal lymphoid target cells. Lymphoid cells positive or negative for a particular specificity were used as controls. The absorption of anti-H-2 sera and the ability of the Meth-A-vaccinia cells to inhibit antibody-complement cytotoxicity on normal lymphoid target cells showed that foreign H-2 antigens on Meth-A-vaccinia tumor cells are expressed. The presence of H-2 specificities on Meth-A and Meth-A vaccinia was also tested by indirect immunofluorescence. The reactivity of these sera was compared using anti-vaccinia virus sera. The unstained noninfected Meth-A and other tumor cells of different H-2 origin. After absorption of these sera on normal or Meth-A tumor cells, there was little or no

activity remaining against Meth-A vaccinia. Tumor growth in syngeneic mice with or without vaccinia virus was also compared. There was inhibition of tumor growth proportional to the doses of virus, the frequency of administration, and the number of Meth-A cells injected. The virus had no effect on the growth of P815Y mastocytoma cells in DBA/2 mice. The results indicate the importance of the expression of foreign H-2 gene products and point to the possibility of immunopotentiality of the host against tumor growth. (23 refs.)

77-2732 **Emergence of Foreign H-2-like Cytotoxicity and Transplantation Targets on Vaccinia and Moloney Virus-infected Meth. A Tumour Cells.** (Eng.) Matossian-Rogers, A. (Tissue Immunology Unit, London Hosp. Medical Coll., Turner St., London E1 2AD, England) Garrido, F.; Festenstein, H. *Scand J Immunol* 6(5): 541-546; 1977.

Cytotoxicity assays with effector cells of different anti-H-2 specificities suggested that new targets for cell-mediated lympholysis (CML) emerged on a methylcholanthrene-induced tumor (Meth.A, H-2d) after passage with vaccinia or Moloney virus. The H-2d CML targets on Meth.A cells recognized by B10.BR anti-B10.D2 effector cells appeared only after simultaneous vaccinia virus passage, but passage with Moloney virus caused the emergence of H-2b targets. Small but significant killing of vaccinia virus-passaged Meth.A was also obtained by anti-H-2k effector cells. These results are discussed in relation to in vivo experiments. Retardation of tumor growth was noted in mice that had received several injections of vaccinia or Moloney virus, showing that the new CML targets were probably acting as transplantation targets. (12 refs.)

77-2733 **Inhibition of Simian Virus 40-Induced Tumors by Antisera to Fetal Hamster Tissue.** (Eng.) Becker, L. E. (Dept. Pathology, Univ. Toronto, Hosp. for Sick Children, 555 University Ave., Toronto, Ontario M5G 1L5, Canada) Narayan, O.; Johnson, R. T. *J Infect Dis* 135(6): 962-964; 1977.

In investigating the protective nature of fetal hamster tissue against infection by simian virus 40 or SV40-transformed tumor cells, it was found that antisera to fetal hamster thymocytes or liver also caused this inhibition. Antisera to adult hamster thymocytes did not have this protective effect. (22 refs.)

77-2734 **Restricted Reactivity of Human Sera to Endogenous Baboon Virus (Meeting Abstract).** (Eng.) Vosika, G. (Univ. Minnesota, Minneapolis, MN 55455) Kennedy, B. J. *Proc Am Assoc Cancer Res* 18: 169; 1977. (no refs.)



- 77-2735 Tumor Immunity in Guinea Pigs: The Chromium-Release Assay as an In Vitro Correlate of Tumor-Host Status and the Effects of Tumor-Bearer Sera on the Assay (Meeting Abstract). (Eng.) Miller, F. R. (Univ. Wisconsin, Madison, WI 53706) *Diss Abstr Int [B]* 38(1): 136; 1977. (no refs.)

- 77-2736 Complement-mediated Antiserum Cytotoxic Reactions to Human Chromosome 7 Coded Antigen(s): Immunoselection of Rearranged Human Chromosome 7 in Human-Mouse Somatic Cell Hybrids. (Eng.) Knowles, B. B. (Wistar Inst. Anatomy and Biology, Philadelphia, PA 19104) Solter, D.; Trinchieri, G.; Maloney, K. M.; Ford, S. R.; Aden, D. P. *J Exp Med* 145(2): 314-326; 1977.

Antisera reactive to cell surface antigen(s) coded for by human chromosome 7 were used to immunoselect clones of human-mouse hybrid cells containing rearranged human chromosome 7. The gene(s) coding for the relevant cell surface antigens in these somatic cell hybrids was localized in the short arm of human chromosome 7. The long arm of the chromosome appeared to contain the simian virus 40 integration site and the gene coding for human  $\beta$ -glucuronidase. Rearrangements of chromosomes retained by stringent selection in somatic cell hybrids can be obtained in hybrid cells if cell surface antigens coded for by the same chromosome exist. (14 refs.)

- 77-2737 Evaluation of the Immune Response in Regional Lymph Nodes of A/J Mice Bearing an Isogenic Tumor, Sarcoma 1 (Meeting Abstract). (Eng.) Barna, B. P. (Cleveland Clinic, Cleveland, OH 44106) Deodhar, S. D. *Fed Proc* 36(3): 1222; 1977. (no refs.)

- 77-2738 Cell Kinetics and Immunogenicity of Lymphoma Cells Treated with 5-(3,3-Dimethyl-1-triazeno) Imidazole-4-carboxamide (DIC) In Vivo. (Eng.) Silvestrini, R. (Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy) Testorelli, C.; Goldin, A.; Nicolin, A. *Int J Cancer* 19(5): 664-669; 1977.

The cell cycle kinetics of the parental and DIC-altered leukemic cells was evaluated by the establishment of labeled mitosis curves. The in vitro DNA synthesis and cell loss were also investigated. The results indicate no significant differences in the behavior of the parental and corresponding drug-treated leukemic sublines. Immunodepressed allogeneic CDF<sub>1</sub> mice were more resistant to lymphoma challenge when inoculated with the DIC sublines than with the parental lines. Upon adoptive transfer of immune lymphocytes, there was increased survival of allogeneic animals challenged with DIC cells. This was attributable to an additional immune response

to DIC-induced antigens. Thus, parental or DIC tumors showed similar tumorigenic characteristics, and the increased allogeneic host survival to DIC cell challenge may be attributed to an additional immune response of the animal against DIC-induced antigens. (21 refs.)

- 77-2739 Cell Surface Components of Carcinogen-Induced Lymphoid Tumors in SJL/J Mice. (Eng.) Haimovich, J. (Dept. Chemical Immunology, Weizmann Inst. Science, Rehovot, Israel) Bergman, Y.; Linker-Israeli, M.; Haran-Ghera, N. *Eur J Immunol* 7: 226-230; 1977.

It was shown that bone marrow (BM) cells from 7,12-dimethylbenz(a)anthracene (DMBA)-induced leukemic SJL/J mice could be evaluated for leukemogenic capacity by iv transplantation of their BM cells into syngeneic recipient mice, and the thymocytes from the leukemic recipient mice differed from those of the donor mice in their antigen response. A 1% solution of DMBA in polyethylene glycol was fed by stomach tube at four weekly intervals, and the BM-containing preleukemic cells were removed and injected iv into recipient mice. The antigenic response was evaluated via assays for thy-1-alloantigen, immunoglobulin G (IgG), and fragment complement (Fc) receptor sites. None of the tumors possessed immunoglobins on their cell surface. Most of the tumors that developed from transplanted DMBA-induced preleukemic BM were found to have Fc receptors, and the original DMBA-induced leukemias did not. All tumor cells formed erythrocyte-antibody rosettes to some extent, and neuraminidase enhanced rosette formation, indicating that some leukemic cell receptor sites on the cell membrane were masked with sialic acid. (21 refs.)

- 77-2740 Induction of Antibodies Against Polycyclic Aromatic Hydrocarbon Carcinogens (Meeting Abstract). (Eng.) Moolten, F. (Boston Univ. Sch. Medicine, Boston, MA 02118) Capparell, N.; Mahathalang, P.; Boger, E. *Proc Am Assoc Cancer Res* 18: 53; 1977. (no refs.)

- 77-2741 Immunization Against MCA Bladder Tumor Development in the Rat (Meeting Abstract). (Eng.) Chapman, W. H. (Dept. Urology, Univ. Washington, Seattle, WA 98195) Wahl, D. V.; Hellstrom, I.; Hellstrom, K. E. *Proc Am Assoc Cancer Res* 18: 159; 1977. (no refs.)

- 77-2742 Characterization of the Tumor Associated Host Cellular Response to Primary 3-Methylcholanthrene Induced Murine Fibrosarcomas (Meeting Abstract). (Eng.) Gollahon, K. A. (Univ. Kansas Medical Center, Kansas City, KS 66103) Wood, G. W. *Proc Am Assoc Cancer Res* 18: 144; 1977. (no refs.)

7-2743 Cell Mediated Immunity to Solubilized Antigens of a Methylcholanthrene (MCA) Induced Fibrosarcoma (Meeting Abstract). (Eng.) Pellis, N. R. Northwestern Univ. Medical Sch., Chicago, IL 60611) Mokyr, M. B.; Kahan, B. D. *Proc Am Assoc Cancer Res* 18: 138; 1977. no refs.)

7-2744 Increased Resistance in Splenectomized Mice to a Methylcholanthrene-induced Tumour. (Eng.) Zhang, R. W. (Dept. Pathology, Royal Coll. Surgeons England, Lincoln's Inn Fields, London WC2A 3PN, England) Turk, J. L. *Br J Cancer* 35(6): 768-776; 1977.

Prior splenectomy increased the resistance of BALB/c mice to a syngeneic methylcholanthrene-induced ascitic tumor inoculated ip. The survival rate of splenectomized mice was 31.6%, but that of normal and sham-operated controls was 1.5% and 20%, respectively. The effect of splenectomy, however, was seen only within the dose range of  $10^3$  to  $10^4$  tumor cells. It was abolished by the transfer to mice of serum from tumor-bearing mice and of spleen cells from normal donors, immediately after tumor cell inoculation. Cell-free ascitic fluid did not abolish the effect of splenectomy. The findings suggest that there is a subpopulation of spleen cells that produces a tumor growth-enhancing factor in the serum of tumor-bearing mice. (5 refs.)

7-2745 An Analysis of the Factors Allowing Promotion (Rather than Inhibition) of Tumour Growth by *Corynebacterium parvum*. (Eng.) Bomford, R. (Dept. Experimental Immunobiology, Wellcome Res. Labs., Beckenham, Kent BR3 3BS, England) *Int J Cancer* 9(5): 673-679; 1977.

Several factors were found to determine whether *Corynebacterium parvum* (CP) treatment promoted rather than inhibited the growth of methylcholanthrene induced fibrosarcoma cells injected into CBA mice. The dose of tumor cells is a significant factor, as promotion occurred only with low doses, around the TD50. The route of injection of CP is also important: greater promotion was caused by iv than by sc administration. Addition of irradiated tumor cells to CP resulted in tumor inhibition. The dose of CP is the third factor--promotion increased with increasing dose of either sc or iv CP. The time of CP injection relative to tumor challenge is the fourth factor. Promotion occurred only when CP was given before the tumor cells, except when iv CP and very few tumor cells were used. With increasing doses of tumor cells, first posttreatment with iv CP and then pretreatment became inhibitory. The effect of CP on established immunity to tumor cells was also studied. Mice were immunized by tumor implantation. The resistance to tumor challenge thus generated could be abrogated by CP given before challenge, most effectively by a high dose iv. The data are interpreted according to the following hypotheses: (1) CP suppresses the expression of cell-mediated immunity to tumor antigens, (2) this is

caused by trapping of antitumor effector cells at the site of CP deposition, (3) promotion can occur only when CP is given before effector cells have reached the tumor site. (39 refs.)

77-2746 Immunological Tolerance and Tumour Allografts in the Brain. (Eng.) Hasek, M. (Inst. Molecular Genetics, Czechoslovak Acad. Sciences, Flemingovo nam. 2, 166 10 Prague 6, Czechoslovakia) Chutna, J.; Sladeczek, M.; Lodin, Z. *Nature* 268(5615): 68-69; 1977.

Inoculation of tumor cells into Lewis rat brains demonstrated transplantation tolerance in the brain, indicating that specific immune processes are accomplished in the CNS. Thus, previous suggestions that the brain is an immunologically privileged site may be incorrect. (8 refs.)

77-2747 Effects of Feeding Syngeneic and Allogeneic Tissues on Growth of an Allotransplantable Tumor. (Eng.) Breyere, E. J. (Dept. Biology, American Univ., Sibley Memorial Hosp., Washington, DC 20016) Heath, J. R. *Transplant Proc* 19(2): 1459-1463; 1977.

The effect of consumption of neonatal syngeneic and allogeneic tissues on the response of adult BALB/c mice to an allotransplantable sarcoma (DBA49) was investigated. Newborns of several strains were fed to BALB/c mice that had been fasted overnight. The mice were later inoculated with the sarcoma (0.025 ml sc), and tumor growth rate was used to indicate host response. Compared with unfed controls, nonspecific increased resistance (decreased growth) was observed in mice fed tissues (1-3 g) of either BALB/c, C3H, or C57BL strains. Feeding allogeneic tissues specifically related to the tumor, ie, DBA/2eB or (C x D)<sub>F1</sub>, resulted in a growth increase over that of nonspecifically fed mice, but one below that of unfed controls. Nonspecific and specific effects reached significant levels in mice inoculated with the tumor 2 and 3 wk after feeding, respectively. Nonspecific resistance was not induced by tissues exposed to 56 C or repeated freezing and thawing, but the specific effect of decreased resistance remained. The results demonstrate that feeding nonspecific allogeneic or syngeneic tissues increases the resistance of murine hosts to an allograft. Specific allogeneic tissues also elicit this response plus an antagonistic effect in the direction of tolerance. (17 refs.)

77-2748 The Systemic Effects of BCG Cell Walls on the Growth of Transplantable Morris Hepatomas in Normal and Splenectomized Rats (Meeting Abstract). (Eng.) Tracey, R. S. (Veterans Admin. Hosp., San Diego, CA, 92161) *Proc Am Assoc Cancer Res* 18: 143; 1977. (1 ref.)



77-2749 Mechanisms of Carcinogenesis (Report 7): Relationship Between Adrenal Corticoidogenesis and Effects of Splenectomy on Implanted Tumor-Development in Rats (Meeting Abstract). (Eng.) Masubuchi, Y. (Dept. Pharmacology, St. Marianna Univ. Sch. Medicine, Kawasaki, 213, Japan) Tanaka, Y.; Yasumuro, K.; Hirai, M. *Jpn J Pharmacol* 26(Suppl): 117P; 1976. (no refs.)

77-2750 Immunological Aspects of Hepatocellular Carcinoma in the Guinea-Pig. (Eng.) Desai, H. N. (Dept. Medicine, Univ. Natal, Durban, Republic South Africa) *S Afr Med J* 50(46): 1863-1866; 1976.

Hepatocellular carcinoma was induced by diethylnitrosamine and propagated in Hartley outbred and Heston inbred guinea pigs, and the antigenicity of the tumor cells was investigated by the macrophage migration inhibition test. The effect of hepatoma VII:3 tumor cells on peritoneal exudate cell (PEC) migration from a sensitized and a normal guinea pig was determined. Increasing the number of tumor cells resulted in an increasing migration inhibition of PECs from the sensitized guinea pig. The PEC migration from the normal guinea pig was stimulated. Experiments with an allogeneic tumor in outbred guinea pigs and with a syngeneic tumor in inbred guinea pigs were carried out. There was a clear-cut dose-dependent migration inhibition of PECs from sensitized guinea pigs with tumor cells. Concomitant measurements were made with the same material on matched control guinea pigs. In virtually all cases, no inhibition was observed. In two outbred guinea pigs, the control unsensitized cells demonstrated some inhibition of migration in the presence of tumor cells. However, the corresponding sensitized cells showed a more significant inhibition. The influence of both VII:3 and XIII:4 tumor cells on PECs from guinea pigs sensitized to one or the other of these two cell lines was determined. The VII:3 and XIII:4 tumor cells did not inhibit the migration of the PECs from unsensitized control guinea pigs measured concomitantly. In the outbred guinea pigs that had been sensitized to tumor VII:3 only, the VII:3 cells produced migration inhibition, but XIII:4 cells did not. In the inbred guinea pigs sensitized to tumor XIII:4, the XIII:4 cells produced migration inhibition, but the VII:3 did not. In the control guinea pigs, the XIII:4 tumor cells produced no migration inhibition, and the normal liver cells produced only a minor degree of migration inhibition. In the sensitized guinea pigs, the XIII:4 tumor cells produced significant inhibition. Carbon tetrachloride-damaged liver cells produced migration inhibition on both normal and sensitized PECs when mixed in ratios above 1:100, whereas XIII:4 tumor cells produced inhibition of sensitized cells only. The degree of inhibition produced by the tumor cells was greater than that caused by chemically damaged cells. At a ratio of 1:100, fetal liver cells did not produce migration inhibition of the sensitized PECs, but the VII:3 tumor cells produced the expected inhibition. The possibility exists of utilizing immunological techniques to study the antigenicity of human cancer. (17 refs.)

77-2751 The Identification of Infiltrating Host Cell Types in Rat Tumours of Varying Immunogenicity (Meeting Abstract). (Eng.) Moore, K. (Paterson Lab., Christie Hosp. and Holt Radium Inst., Manchester M20 9BX, England) Moore, M. *Br J Cancer* 35(2): 257; 1977. (no refs.)

77-2752 Depression of Mononuclear Phagocyte Function by Lewis Lung Carcinoma in C57BL Mice (Meeting Abstract). (Eng.) Otu, A. A. (Univ. Dept. Bacteriology and Immunology, Western Infirmary, Glasgow W1 Scotland) Russell, R. J.; Wilkinson, P. C. *Br J Cancer* 35(2): 252; 1977. (2 refs.)

77-2753 Suppression of Macrophage-Mediated Immunity to Leukemia L1210 by Immune Complexes (Meeting Abstract). (Eng.) Rao, V. S. (Dept. Medicine and Pharmacology, Yale Univ. Sch. Medicine, New Haven, CT 06510) Mitchell, M. S. *Proc Am Assoc Cancer Res* 18: 63; 1977. (no refs.)

77-2754 Presence of Splenic Suppressor Cells in Mice Bearing Regressively Growing Moloney Sarcomas and Their Absence in Progressor Mice. (Eng.) Weiland, E. (Federal Res. Inst. for Animal Virus Diseases, Tübingen, W. Germany) Mussgay, M. *Eur J Cancer* 13(7): 705-711; 1977.

Cultured spleen cells from STU inbred mice inoculated with Moloney murine sarcoma virus (MSV-M)-induced ascites tumor cells ( $10^5$  and  $10^6$  cells) were examined for their responsiveness to mitogens at the beginning of tumor development (6 days posttransplantation, the peak phase (13 days), and the regression period (18 days). Spleen cells from mice at the stage of peak tumor size did not show induction of blastogenesis by concanavalin A and phytohemagglutinin as measured by  $^3\text{H}$ -thymidine uptake. Moreover, the spontaneous  $^3\text{H}$ -thymidine uptake was markedly depressed in the presence of the mitogens, especially phytohemagglutinin. However, the concomitantly assessed activity of these spleen cells in an  $^3\text{H}$ -proline microcytotoxicity assay was strong. The depression of the mitogen response restricted to the peak tumor phase; it was not observed during tumor development and regression. Spleen cells of mice bearing a nonregressing tumor with a development comparable with that of the Moloney sarcoma in the progression stage did not show a depression of mitogen response and had no cytotoxic activity. Therefore, the absence of detectable cytotoxic effector cells was not due to the activity of suppressor cells, and the tumor growth rate had no influence on the development of the suppressor cells. (13 refs.)

7-2755 Specific Suppressor Cells in Mice Bearing a Syngeneic Mastocytoma (Meeting Abstract). (Eng.) Takei, F. (Univ. British Columbia, Vancouver, British Columbia, Canada) *Diss Abstr Int [B]* 38(1): 137; 1977. (no refs.)

7-2756 Tumor-Specific Suppressor Cells Induced by Ultraviolet (UV) Light (Meeting Abstract). (Eng.) Fisher, M. S. (NCI-Frederick Cancer Res. Center, Frederick, MD 21701) *Proc Am Assoc Cancer Res* 18: 55; 1977. (no refs.)

7-2757 Influence of Immunosuppression on Experimental Induction of Brain Tumors in the Rat (Meeting Abstract). (Ger.) Batka, H. (Erfurt, E. Germany) *Wartburg, R.; Scholtze, P. Zentralbl Allg Pathol* 121(3): 247-275; 1977. (no refs.)

7-2758 Immunosuppressive Proteins in Breast Carcinoma (Letter to Editor). (Eng.) Virtue, C. M. (Madigan Army Medical Center, Tacoma, WA 98431) *Oppe, A. N Engl J Med* 296(24): 1412; 1977.

The recovery of immunosuppressive proteins from breast carcinoma tissue is reported. The saline-soluble proteins were found in the first recovery fraction on G-200 Sephadex separation, migrated in the A<sup>2</sup> region on electrophoresis, and markedly inhibited leukocyte response suppressive proteins without responding to their suppressive function suggests that malignant transformation may involve a block at DNA receptor sites for regulatory proteins. (no refs.)

7-2759 Immunosuppressive Properties of Tobacco Smoke Condensate (Meeting Abstract). (Eng.) Jacob, C. V. (Univ. Louisville, Louisville, KY 40208) *Diss Abstr Int [B]* 37(8): 3780; 1977. (no refs.)

7-2760 Immunosuppressive Properties of AFP: Role of Estrogens. (Eng.) Keller, R. H.; Calvanico, N. Tomasi, T. B. In: *Onco-Developmental Gene Expression*. Shiman, W. H.; Sell, S., eds. (New York: Academic Press, Inc.): pp. 287-295; 1976.

Fetoprotein (AFP) isolated from mouse (Ha/ICR) amniotic fluid was immunosuppressive only when complexed with an appropriate smaller molecule, probably an estrogen closely related to estradiol. The AFP was isolated on estradiol affinity columns. AFP depleted of estrogens was nonsuppressive and estradiol partly restored the suppressive properties. Estrone, which binds tightly to AFP, did not restore suppression. It is possible that there are two forms of AFP, one an

estrogen binder that is immunosuppressive, and another that is a nonbinder and nonsuppressive. The estrogen-AFP complex may act via an allosteric effect producing a suppression of AFP molecule. Some of the discrepancies in the literature regarding the suppressive properties of AFP from various sources (serum, hepatoma, amniotic fluid, etc.) and differences between species could be explained either on the basis of their content of AFP molecules having different abilities to bind estrogens or the lack of an appropriate activating estrogen. (15 refs.)

77-2761 Fetal Antigen Modulation in Chemical and Viral Transformed Cells (Meeting Abstract). (Eng.) Evans, D. L. (Bowman Gray Sch. Medicine, Winston-Salem, NC 27103) *Parker, D. V. In Vitro* 13(3): 171; 1977. (no refs.)

77-2762 Separation of the Complement-fixing and Early Antigens from Epstein-Barr Soluble Antigen. (Eng.) Wainwright, W. H. (Dept. Microbiology, West Virginia Univ. Medical Center, Morgantown, WV 26506) *Veltri, R. W. J Natl Cancer Inst* 58(4): 1111-1113; 1977.

The Epstein-Barr virus associated complement-fixing soluble antigen was isolated from 5'-iodo-2'-deoxyuridine activated P3HR-1 lymphoblastoid cells and from RAJI cells. Early antigen was also isolated from the former, but not the latter. These antigens had different immunological properties. (12 refs.)

77-2763 Cell Surface Antigens Detected in Cell Lines Established from Lymphomatous *Papio hamadryas* and *Macaca arctoides* Monkeys. (Eng.) Bubenik, J. (Inst. Experimental Biology and Genetics, Czechoslovak Acad. Sciences, 160 00 Prague 6, Czechoslovakia) *Jandlova, T.; Simova, J.; Agrba, V. Z.; Yakovleva, J. A.; Lapin, B. A.; Kokoscha, L. V.; Klepikov, N. N.; Voevodin, V. F. Neoplasma* 23(5): 463-470; 1977.

Cell surface antigens in cultures of bone marrow cells from leukemic *Papio hamadryas* and *Macacus arctoides* monkeys were visualized by indirect immunofluorescence using sera from leukemic baboons. The same immune serum gave two types of immunofluorescence, depending on the origin of the target cells. Ring-reaction fluorescence was seen with *P. hamadryas* bone marrow cell cultures growing in suspension and containing baboon herpesvirus, but patchy fluorescence was noted in monolayer bone marrow cell cultures derived from *M. arctoides* and containing C-type oncornavirus particles. The antibodies responsible for the patchy fluorescence could be absorbed with a disintegrated C-type baboon oncornavirus, but not with baboon lymphoblastoid cell lines containing herpesvirus or with human lymphoblastoid lines containing Epstein-Barr virus. (21 refs.)



- 77-2764 Cell Surface Antigens of Totipotent Mouse Teratocarcinoma Cells Grown In Vivo: Their Relation to Embryo, Adult, and Tumor Antigens.** (Eng.) Dewey, M. J. (Inst. Cancer Res., Fox Chase, Philadelphia, PA 19111) Gearhart, J. D.; Mintz, B. *Dev Biol* 55(2): 359-374; 1977.

The cell surface antigens on mouse embryonal carcinoma or teratocarcinoma cells were assessed by a syngeneic antiserum prepared against small-size embryoid bodies from the ascites form of the OTT 6050 transplantable teratoma. According to indirect immunofluorescence, the antiserum prepared in strain 129 mice against small embryoid bodies and naked cores reacted with the surfaces of embryonal carcinoma cells dissected as plugs from small embryoid bodies, after mild proteolysis had loosened the yolk sac epithelial covering and enabled it to be stripped off completely. The antiserum was unreactive against unfertilized eggs but reacted positively with all the early mouse embryonic stages (two-cell stage up through the late day 6 egg cylinder). Reactivity was weakest in two-cell (day 1) embryos and increased in intensity as cleavage progressed on day 2. Assay of day 3 blastocysts revealed a weak reaction on trophoblasts and a more intense membrane fluorescence on cells of the inner cell mass. There was a striking persistence of reactivity to at least the late day 6 egg cylinder stage, with the ectoderm and endoderm of the embryo displaying specific membrane fluorescence. Day 2 embryos with close to 10 cells each were collected from  $+ / t^{12} \times + / t^{12}$  matings and assayed for surface reactivity. Of a total of 56 embryos tested, all fluoresced positively, with comparable intensity. The antiserum failed to react with strain 129 thymocytes or fibroblasts in primary cultures from fetuses or with an established line of 3T3 fibroblasts. Approx half the reactivity against the embryoid body yolk sac epithelium was removed by absorption with a mixture of adult brain, liver, kidney, and spleen tissue of the mice. Almost all reactivity to this epithelium was removed by prior absorption with ovary or testis tissue. No evidence is adduced to support the hypothesis that surface components required for normal early development are coded by the wild-type allele of  $t^{12}$ . (37 refs.)

- 77-2765 Antibody-induced Redistribution of Marek's Disease Tumor-associated Surface Antigen (MATSA) on Lymphoblastoid Line (MSB-1) Cells Derived from Marek's Disease Lymphoma.** (Eng.) Matsuda, H. (Dept. Pathology, Res. Inst. Microbial Diseases, Osaka Univ., Yamada-kami, Suita, Osaka, Japan) Ikuta, K.; Miyamoto, H.; Kato, S. *Biken J* 20(1): 35-37; 1977.

Using sera of rabbits and chickens immunized with Marek's disease lymphoblastosis MSB-1 line cells, it was shown that Marek's disease tumor-associated surface antigen (MATSA) was present on cells from all six Marek's disease lymphoblastoid cell lines. Reactions with rabbit anti-MSB-1 serum demonstrated temperature-dependent polarization, cap-

formation, and cap-exclusion phenomenon of MATSA determinants on the surface of MSB-1 cells. The movement of MATSA on or in the membrane may be important in the relationship of the membrane tumor-specific antigen to immune expression. (7 refs.)

- 77-2766 Antibody-induced Antigen Redistribution and Shedding from Human Breast Cancer Cells.** (Eng.) Nordquist, R. E. (Cancer Res. Program, Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104) Anglin, J. H.; Lerner, M. P. *Science* 197(4301): 366-367; 1977.

In vitro studies with BOT-2 human breast cancer cells and antibodies to these cells indicated that cell surface antigens from the cancer undergo redistribution upon antibody binding and subsequent shedding of these antigen-antibody complexes and free antigen. These antigens compete with the tumor for the immune response, and this may enhance tumor survival. (8 refs.)

- 77-2767 Immunogenicity of Solubilized Tumor Antigen Extracted from P1798 Murine Lymphoma Cells or Isolated from Tumor-bearer Ascites Fluid and Reactivity with Anti-Thy-1.2 Antiserum.** (Eng.) Gordon, W. C. (Dept. Surgery, Univ. Texas Health Science Center at Dallas, Dallas, TX 75235) Baechtel, F. S.; Goetz, G.; Prager, M. D. *Int J Cancer* 19(5): 692-699; 1977.

Solubilized antigen was prepared from P1798 lymphoma cells by sonication or 3M KCl extraction, or it was isolated from the ascites fluid of syngeneic tumor-bearing BALB/c mice. Antigen was detected and quantitated by its ability to block the activity of anti-P1798 serum raised in syngeneic mice, as assayed by cytotoxicity and indirect immunofluorescence tests. The reaction was found to be immunologically specific, as the P1798 antigen did not inhibit the binding to L1210 lymphoma cells of antisera raised against L1210 in syngeneic DBA/2 or allogeneic BALB/c mice. Ip vaccination of BALB/c mice with different subcellular fractions of sonicated antigen or with ascites fluid protected them against live P1798 challenge ( $10^3$  cells), with results comparable to those obtained using iodoacetamide-modified tumor cells. Solubilized antigen prepared by each of the three methods eluted from a Bio-Gel A5m agarose column exclusively in an early peak that had a molecular wt  $> 2 \times 10^6$ . This column-fractionated antigen cross-reacted with antiserum raised against Thy-1.2 antigen, which is present on P1798 cells. The purified P1798 antigen sedimented at 200,000 g and protected syngeneic mice in immunoprophylactic tests. (31 refs.)

- 77-2768 Studies of Tumor Specific Antigens on Leukemic Cells by Mixed Lymphocyte-Leukemic Cell Cultures.** (Jpn.) Ohno, R. (First Dept. Internal Medicine, Nagoya Univ. Sch. Medicine, Nagoya, Japan) Morishima,

; Kato, Y.; Sugiura, S.; Takeyama, H.; Wakayama, K.; Jeda, R.; Ezaki, K.; Yamada, K. *Acta Haematol Jpn* 40(2): 77-182; 1977.

An attempt was made to detect tumor-specific antigens on acute leukemia cells in mixed cultures of lymphocytes and autochthonous leukemia cells. The lymphocytes used were obtained from patients in chemotherapy-induced complete remission. The leukemia cells had been taken from the peripheral blood or bone marrow of leukemia patients before chemotherapy and then preserved in liquid nitrogen in medium supplemented with 10% dimethyl sulfoxide and 10% fetal calf serum until use. The ratios of lymphocytes to leukemia cells (untreated or irradiated with 4,000 R) were 1:1 or 1:2, and the mixtures were cultured for 7 days in RPMI 1640 medium supplemented with 20% fresh human AB serum at 37°C in 5% CO<sub>2</sub> in air. Among 14 cases of acute leukemia in remission, the lymphocytes of 8 showed a significant blastoid response to the preserved autochthonous leukemia cells. In all six cases with peroxidase-positive leukemia cells showed significant response, as opposed to 2/8 cases with peroxidase-negative leukemia cells. Normal bone marrow cells preserved by the same process did not stimulate autochthonous lymphocytes in the mixed cultures. Positive responses were seen in 4/6 patients with acute myeloblastic leukemia, 2/2 with acute monoblastic leukemia, 1/1 with erythroleukemia, and 1/5 with acute lymphoblastic leukemia. (20 refs.)

77-2769 **Partial Serological and Biochemical Characterization of Human Melanoma Tumor Associated Antigens (Meeting Abstract).** (Eng.) Stuhlmiller, G. M. (Duke Univ., Durham, NC 27706) *Diss Abstr Int [B]* 37(12/1): 6060-6051; 1977. (no refs.)

77-2770 **HLA Antigens in Malignant Melanoma.** (Eng.) Bergholtz, B. (Tissue Typing Lab., Natl. Hosp. Norway, Oslo, Norway) Brennhovd, I.; Klepp, O.; Kaakinen, E.; Thorsby, E. *Cancer* 39(6): 2342-2344; 1977.

HLA typing of 54 patients with malignant melanoma indicated a slight, but nonsignificant increase in the frequency of HLA-B27 and LD108 in the patients as compared to healthy controls. (15 refs.)

77-2771 **Frequency of HLA Antigens in Chronic Myelocytic Leukemia.** (Eng.) Hester, J. P. (Dept. Developmental Therapeutics, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) Rossen, R.; Trujillo, J.; McCredie, K. B.; Freireich, E. J. *South Med J* 70(6): 691-693; 1977.

HLA phenotypes of 43 patients with Philadelphia chromosome-positive chronic myelogenous leukemia (CML) were evaluated for association with

HLA antigens. Two control populations were compared with the CML patients: 142 normal volunteer platelet donors and 160 normal donors of granulocyte transfusions. HLA typing was done by lymphocyte microcytotoxicity tests for 9 antigens on sublocus A and 15 antigens on sublocus B. Compared with the controls, the CML patients showed an increased frequency of HLA-B5, A11, B8, and BW17 and a decreased frequency of HLA-B12, B7, and BW35. The median survival was 24+ mo (range 4+ to 84+ mo), and it was independent of HLA. The precise relationship of HLA to disease states remains to be clarified. (15 refs.)

77-2772 **Studies on Cellular Inhibition and Serum-blocking Factors in 28 Human Patients Given Marrow Grafts from HLA Identical Siblings.** (Eng.) Tsoi, M. S. (Fred Hutchinson Cancer Res. Center, 1124 Columbia St., Seattle, WA 98104) Storb, R.; Weiden, P. L.; Thomas, E. D. *J Immunol* 118(5): 1799-1805; 1977.

Fifteen patients with aplastic anemia and 13 with acute leukemia were investigated 36-1,547 days following treatment with high-dose cyclophosphamide and/or total-body irradiation and marrow transplantation from histocompatibility antigen (HLA)-identical siblings. Fourteen patients were tested between 250 and 1,547 days postgrafting, 12 on one and 2 on two occasions. Eleven of these patients had no cell inhibition (CI) and, hence, no serum blocking, but 3 had both specific CI and serum-blocking activity. These patients were stable long-term survivors without evidence of graft-vs-host disease (GVHD). Ten short-term survivors without acute GVHD between 36 and 144 days postgrafting demonstrated CI without blocking on three occasions, CI with blocking on four occasions, and the absence of both on three occasions. Two short-term survivors with acute GVHD between 36 and 144 days postgrafting demonstrated CI and blocking on three occasions and the absence of CI on one occasion. Two patients who had chronic GVHD either at the time of testing or who developed chronic GVHD after CI testing between days 61 and 960 postgrafting demonstrated CI without blocking on two occasions and absence of CI and blocking on seven. The maintenance of GVH tolerance in long-term survivors following marrow grafting from antigen identical donors does not depend on serum-blocking factors. (28 refs.)

77-2773 **Autoimmunization and Lymphomagenesis in Parent→F<sub>1</sub> Hybrid Combinations Differing at the Major Histocompatibility Complex (MHC) (Meeting Abstract).** (Eng.) Gleichmann, E. (Div. Experimental Pathology, Hannover, W. Germany) Gleichmann, H. *Scand J Immunol* 6(6/7): 701-702; 1977. (no refs.)

77-2774 **Sequential Quantitation of Circulating Immune Complexes in Syngeneic and Allogeneic Rats Bearing Moloney Sarcomas.** (Eng.) Jennette, J. C. (Dept. Im-



munopathology, Scripps Clinic and Res. Foundation, La Jolla, CA 92037) Feldman, J. D. *J Immunol* 118(6): 2269-2274; 1977.

A Raji cell radioimmunoassay was used for the serial quantitation of circulating immune complexes (CIC) in the sera of syngeneic BN rats and allogeneic Lewis rats bearing BN Moloney sarcomas. In syngeneic BN hosts, the CIC levels attained and the time course of detection were related to the tumor dose, tumor mass, and regressive or progressive course of the tumor. In general, syngeneic rats that received larger tumor doses developed larger tumors and greater max levels of CIC. The amount of CIC was not always directly proportional to tumor size, although this was the case with most regressor BN and Lewis rats. In rats with regressing tumors, CIC decreased to insignificant levels as the tumors disappeared. Progressor BN rats that received 20 and 10 x 10<sup>6</sup> tumor cells had higher and more sustained levels of CIC, but the levels declined shortly before the terminal phase of tumor progression. Progressor BN rats that received an initial inoculum of 0.5 x 10<sup>6</sup> tumor cells that grew to 44 mm max mean diameter had CIC levels only slightly above those of control rats. All allogeneic Lewis hosts rejected the BN Moloney sarcomas, but they had transient low levels of CIC coincident with tumor growth. Lewis rats had lower levels of CIC than BN rats bearing comparable masses of sarcoma. (36 refs.)

- 77-2775 **Mechanisms of the H-2 Effect on Viral Leukemogenesis.** (Eng.) Bubbers, J. E. (Dept. Genetics, Albert Einstein Coll. Medicine, Bronx, NY 10461) Blank, K. J.; Freedman, H. A.; Lilly, F. *Scand J Immunol* 6(5): 533-534; 1977.

Studies of the effect of the H-2 haplotype on the immune response of congenic BALB mice to Friend murine leukemia virus (FV)-induced tumors are reviewed. The results support the notion that two distinct but interacting mechanisms, controlled by loci mapping within the H-2 complex, influence FV disease. One mechanism, controlled by a gene mapping in or close to H-2D, influences the capacity of the H-2D gene product to form molecular complexes with FV molecules in the plasma membrane of infected cells. Complex formation appears to provide a target antigen for syngeneic cytotoxic T lymphocytes, to cause cocapping of FV and H-2D antigens, to permit the selective inclusion of H-2D molecules into progeny Friend virions, to influence the long-term maintenance of virus production in vitro, and, in conjunction with the second mechanism, to stimulate the generation of cytotoxic T-lymphocytes. This second mechanism is controlled by a gene in the H-2K or H-2I region, and, in the presence of an H-2/FV molecular complex immunogen, influences the generation of H-2 restricted cytotoxic T lymphocytes and the rate of rejection of syngeneic FV-induced tumor cells. (16 refs.)

- 77-2776 **Helper Antigen Augmentation of Tumor Transplantation Antigen Activity Using Influenza and Vesicular Stomatitis Viruses.** (Eng.) Boone, C. W.; Gillette, R. W. In: *Modulation of Host Immune Resistance in the Prevention or Treatment of Induced Neoplasias*. NCI and John E. Fogarty International Center for Advanced Study in the Health Sciences. (Bethesda, MD): Fogarty International Center Proceedings No. 28 pp. 191-194; 1977.

Cultured monolayers of E<sub>4</sub> mouse fibrosarcoma cells were infected with either the WSN strain of mouse-adapted influenza virus or vesicular stomatitis virus (VSV). When tumor-immune mice were challenged sc with 0.03 ml of tumor homogenate (10<sup>6</sup> tumor cells), augmented immunogenicity was induced. The insoluble material obtained by dialysis of a 3 M KCl extract of influenza virus-infected tumor cells rendered mice highly immune to an sc challenge with 10<sup>6</sup> tumor cells. A radioisotopic footpad assay using <sup>125</sup>I-labeled mouse serum albumin injected ip into tumor-immune mice indicated that VSV-infected cells elicited a strong delayed-hypersensitivity response. The possible significance of these results is discussed. (13 refs.)

- 77-2777 **Intracellular Antigens Expressed in Mammalian Cells with Unlimited Growth Potential in Culture.** (Eng.) Ebersolt, C. (Departement de Biologie moleculaire, Institut Pasteur, Paris, France) Paulin, D.; Cuzin, F. *Biol Cell* 28(2): 109-114; 1977.

An antigenic structure was detected in the perinuclear region of mouse cells (and those of other species) with the aid of an antiserum directed against a crude nuclear fraction from 3T6 mouse fibroblast nuclei. The antigens were expressed in all cell lines tested, including Swiss 3T6, BALB/c and Swiss 3T3, simian virus 40- or polyoma virus-transformed 3T3, and teratocarcinoma cells. They were not detectable in differentiated mouse cells and were barely detectable in primary culture cells. The expression of these antigens appears to be related to acquisition of the immortal phenotype, characterized by the ability to multiply in culture for an indefinite number of generations. Their appearance, as detected by immunoenzymatic staining of fixed cells, was linked to a structural rearrangement of preexisting antigenic sites, rather than to the synthesis of new antigens. (30 refs.)

- 77-2778 **Non-H-2 Foreign Alloantigens on Chemically Induced Sarcoma Cells as Detected by Graft-versus-host Reaction.** (Eng.) Parmiani, G. (Div. Experimental Oncology A, Natl. Cancer Inst., Milano, Italy) Invernizzi, G. *Folia Biol (Praha)* 22(6): 395-396; 1976.

Experimental results are presented which indicate that lymphocytes of BALB/c mice immune to syngeneic ST2 and ST5, but not to an unrelated C-1 fibrosarcoma, behave like BALB/c anti-DBA/2 lymphocytes in that they show a spe-

cific increase in the graft-vs-host reaction in (BALB/c x DBA/2) $F_1$  mice. (11 refs.)

77-2779 **Antigen Expression on Cells of Rat Tumour Xenografts in Athymic Nude Mice (Meeting Abstract).** (Eng.) Pimm, M. V. (Cancer Res. Campaign Lab., Univ. Nottingham, Univ. Park, Nottingham NG7 2RD, England) *Br J Cancer* 35(2): 252-253; 1977. (no refs.)

77-2780 **Expression of Normal Mammary Epithelial Cell Antigens in Mammary Neoplasia (Meeting Abstract).** (Eng.) Ceriani, R. L. (Bruce Lyon Memorial Res. Lab., Children's Hosp. Medical Center, Oakland, CA 94609) Peterson, J. A.; Abraham, S. *Proc Am Assoc Cancer Res* 18: 60; 1977. (no refs.)

77-2781 **Cell Surface Binding Factors (Antibodies?) to X-Irradiation Induced Adenocarcinoma (Meeting Abstract).** (Eng.) Hoffman, K. L. (Radiation Res. Lab., Dept. Radiology, Univ. Iowa, Iowa City, IA 52242) Stevens, R. H.; Brooks, G. P.; Osborne, J. W.; Cheng, H. F. *Radiat Res* 70(3): 689; 1977. (no refs.)

77-2782 **Smooth Muscle Antibody in Burkitt's Lymphoma and in Nasopharyngeal Carcinoma.** (Eng.) Lamelin, J. P. (International Agency Res. on Cancer, 9372 Lyon Cedex 2, France) Williams, E. H.; Souissi, T.; De-The, G.; Gabbiani, G. *Clin Exp Immunol* 28(1): 157-162; 1977.

Sera from 15 Ugandan patients with Burkitt's lymphoma (BL) and 30 Tunisian patients with nasopharyngeal carcinoma (NPC) were assayed for smooth muscle antibodies (SMA) with specificity for actin. The frequency of SMA-positive sera was higher in both patient groups (11/15 and 13/30, respectively) than in matched controls (5/15 and 5/89, respectively). No correlation could be found between SMA and anti-Epstein-Barr virus (EBV) antibody titers. In individual sera, there was no correlation between SMA and the occurrence of cold lymphocytotoxins, another antibody activity found with an abnormally high frequency among BL and NPC patients. The reason why actin, a weak antigen in experimental animals, may become immunogenic in humans remains unexplained. (24 refs.)

77-2783 **Uniformity in a Clonal Repertoire: A Case for a Germ-line Basis of Antibody Diversity.** (Eng.) Laflin, J. L. (Dept. Microbiology, Univ. Michigan Medical Center, Ann Arbor, MI 48019) Rudikoff, S. *Cold Spring Harbor Symp Quant Biol* 41: 725-734; 1977.

To study the question of antibody diversity, the composition of antiphosphorylcholine (anti-PC) antibodies formed in 18 different inbred strains of mice by immunization with *Streptococcus pneumoniae* was examined by quantitative idiotype analysis, structural studies of light (L) chains using analytical isoelectric focusing, and sequence analysis of specific antibodies. Antibodies bearing binding-site structural features of three different BALB/c myeloma proteins, M603, T15, and M511, were regularly expressed, and they comprised the majority of the PC response. Almost all strains produced antibodies with L chains that cofocus with T15 and M511 L chains, and all but BALB/cJ and C58J mice produced M603-like L chains. This uniformity could be extended to the immunoglobulins themselves, indicating that the anti-PC antibodies are similar in composition. The genes coding for L and H (heavy) chains in anti-PC antibodies from both A/J and BALB/c mice, two unrelated strains, are very similar and must be contained in the same germ line. Three to four clones, or a clone for each idiotype, may be responsible for generating antibodies to PC. (48 refs.)

77-2784 **Tumor Growth in the Absence of Circulating Antibodies.** (Eng.) Nowygrod, R. (Dept. Surgery, Columbia-Presbyterian Medical Center, 622 W. 168 St., New York, NY 10032) Sutherland, D. E.; Howard, R. J.; Najarian, J. S. *J Surg Res* 22(6): 660-666; 1977.

The growth of avian myeloblastosis virus-induced nephroblastomas in agammaglobulinemic WC and FS chickens was studied to test the hypothesis that tolerance to tumor antigen is the consequence of an antibody-mediated inhibition of cellular rejection by T cells. Agammaglobulinemia was effected by bursectomy and irradiation (675 R) performed within 24 hr of hatching. Only chickens with undetectable serum immunoglobulin (IgG and IgM) and with negative response to hyperimmunization with *Brucella abortus* and sheep RBC were considered truly bursectomized. Intrastrain nephroblastoma tumor transplants resulted in tumor growth in 14/17 chickens; interstrain growth occurred in 5/11 hosts. Seven of 12 agammaglobulinemic chickens and 12/16 nonagammaglobulinemic chickens developed tumors, suggesting that tumor growth did not depend on antibody-mediated enhancement. The possibility that depressed T-cell function resulting from bursectomy and irradiation influenced tumor development was excluded by the normal skin-graft rejection and graft-vs-host reactivity observed in the agammaglobulinemic chickens. (26 refs.)

77-2785 **Determination of Specificity in Natural Cell Mediated Cytotoxicity by Natural Antibodies (Meeting Abstract).** (Eng.) Koide, Y. (Univ. California, Los Angeles, CA 90024) Takasugi, M. *Proc Am Assoc Cancer Res* 18: 161; 1977. (no refs.)



77-2786 **Antibodies in Human Sera to an Oncornavirus (V-L104) Isolated from Human Tumor Cells Cocultivated with Rat Cells (Meeting Abstract).** (Eng.) Gabelman, N. (Mount Sinai Sch. Medicine, New York, NY 10029) Robinson, A.; Waxman, S. *Proc Am Assoc Cancer Res* 18: 135; 1977. (no refs.)

77-2787 **Humoral Antibodies to the Capsid Antigen of Epstein-Barr Virus in Hodgkin's Disease.** (Eng.) Stepina, V. N. (Oncological Scientific Center, Acad. Medical Sciences USSR, Moscow 115 478, USSR) Gourtsevich, V. E.; Mazurenko, N. P.; Yarymova, N. M.; Kaverznye, M. M.; Lorye, Y. I. *Neoplasma* 23(5): 523-532; 1977.

A study of indirect immunofluorescence reactions between sera from 64 patients with Hodgkin's disease and P3HR-I cells (derived from the P3J strain of Burkitt's lymphoma) showed that the sera contained high titers of humoral antibodies to Epstein-Barr virus (EBV) capsid antigen. The lymphocyte depletion and mixed cellular forms of the disease, which have an unfavorable clinical course, exhibited higher antibody titers than did the nodular-sclerotic form, which has a more favorable clinical course. However, antibody levels were not correlated with the results of therapy. Sera from patients with reticulosarcoma or lymphosarcoma did not have increased EBV antibody titers compared with sera from healthy donors; sera from patients with acute leukemia tended to have decreased levels of EBV antibodies. (45 refs.)

77-2788 **Myeloma Specific Antibodies: Studies on Their Occurrence, Properties, and Their Relationship to Transplantation Immunity (Meeting Abstract).** (Eng.) Frikke, M. J. (Washington Univ., St. Louis, MO 63130) *Diss Abstr Int [B]* 37(8): 3862; 1977. (no refs.)

77-2789 **Primary Structural Differences in Myeloma Proteins That Bind the Same Haptens.** (Eng.) Potter, M. (NCI, Lab. Cell Biology, Bethesda, MD 20014) Rudikoff, S.; Vrana, M.; Rao, D. N.; Mushinski, E. B. *Cold Spring Harbor Symp Quant Biol* 41: 661-666; 1977.

Amino acid sequences of antigen-binding myeloma proteins of BALB/c origin with a specificity for phosphorylcholine (PC), inulin and  $\beta$ -6 D-galactan are presented. The findings indicate that rabbit antisera recognize cross-specific idiotypes common to galactan-binding myeloma proteins and antigalactan antibodies induced in BALB/c mice. It was not possible to determine whether the differences in the three inulin-binding protein variable heavy-chain (VH) sequences are germ-line-carried or somatic mutations. The fact that a series of  $\kappa$ -type myeloma proteins can be retrieved randomly indicates that the cells producing these specificities are expressed regularly and that they are a susceptible population for neoplastic transformation. Similarities in the primary

structure of proteins within a group indicate that specific binding activities in the mouse depend upon activation of specific variable light chain (VL) and VH genes. The V region sequences provide some insights into the genetic control and differentiation of immunoglobulin V genes, whose activation may depend upon enzymes specific for individual or sets of genes. (38 refs.)

77-2790 **Regulation of Immunoglobulin Expression in Mouse Myeloma Cells.** (Eng.) Margulies, D. H. (Dept. Cell Biology, Albert Einstein Coll. Medicine, Bronx, NY 10461) Cieplinski, W.; Dharmgrongartama, B.; Gefter, M. L.; Morrison, S. L.; Kelly, T.; Scharff, M. D. *Cold Spring Harbor Symp Quant Biol* 41: 781-791; 1977.

Cloned mouse myeloma lines were used in genetic and biochemical studies to learn more about the mechanisms that regulate immunoglobulin (Ig) expression. Fusions carried out between different heavy (H) and light (L) chain-producing myeloma cells representing the major classes and subclasses of mouse Ig indicate that genetic expression in differentiated plasma cells is under cis-dominant control. Preliminary results of fusions between parental cells and nonproducing variants also support this type of control. According to complementation analyses, the nonproducing cell-free system does not prevent the translation of H- and L-chain messages. Additional studies indicate that there is no interdependence between homologous H and L chains. However, observations confirm that quantitative modulation of Ig production occurs. Four groups of somatic cell hybridization studies involving Ig-producing cells are reviewed. (54 refs.)

77-2791 **Comparison of Immunoglobulin Chains Made in Ascites Extract and Reticulocyte Lysate Programmed with mRNA from Four Mouse Myelomas.** (Eng.) Schmeckpeper, B. J. (Traylor Res. Building Room 933, Johns Hopkins Univ. Sch. Medicine, 720 Rutland Ave., Baltimore, MD 21205) Cory, S.; Adams, J. M. *Biochim Biophys Acta* 476(4): 303-320; 1977.

The translation of microsomal messenger RNA (mRNA) from four mouse myelomas was studied in a supplemented Krebs II ascites cell extract and in a rabbit reticulocyte lysate to investigate the cell-free synthesis of immunoglobulin (Ig) chains. In the ascites system, translation of Ig light (L)-chain mRNA's was enhanced by the addition of 18S ribosomal RNA, ascites transfer RNA, and KCl concentrations higher than the optimum for total amino acid incorporation. Reticulocyte initiation factors strongly stimulated the translation of exogenous and endogenous mRNA's. The background of endogenous incorporation was not eliminated by preincubation of the extract. mRNA from each of the myelomas directed the synthesis of L chains and their precursors, P chains, in the ascites and lysate systems. The P chains ranged in size from 1,300 to 2,200 daltons larger than the

corresponding L chains. The P chains made in response to a particular mRNA were indistinguishable in size in the ascites extract and in the reticulocyte lysate (and in a previously investigated wheat germ system). Small amounts of large polypeptides serologically related to  $\alpha$  and  $\gamma$  heavy chains were made in the ascites system, and large amounts of an  $\alpha$ -chain-related product were made in the reticulocyte system, probably in a precursor form. These results strengthen the case that P chains are a general feature of Ig L-chain mRNA translation. (40 refs.)

**77-2792 IgA Antibodies to Epstein-Barr Viral Capsid Antigens in Saliva of Nasopharyngeal Carcinoma Patients.** (Eng.) Ho, H. C. (Medical and Health Dept., Inst. Radiology and Oncology, Queen Elizabeth Hosp., Kowloon, Hong Kong) Ng, M. H.; Kwan, H. C. *Br J Cancer* 35(6): 888-890; 1977.

Sera and saliva specimens from 30 patients with nasopharyngeal carcinoma, 20 patients with other cancers, and 10 healthy subjects were tested for the presence of IgA antibodies to Epstein-Barr virus viral capsid antigens (VCA) by immunofluorescence. Staining with fluorescein-conjugated anti- $\alpha$  serum revealed IgA reactivity to VCA in all 30 sera and 24 saliva specimens from the NPC patients. Control specimens were negative. Serum and saliva IgA was detectable by double immunodiffusion with the anti- $\alpha$  serum in all NPC patients and all controls. IgG was present in the saliva of 27 NPC patients, 19 with other cancers, and 9 healthy subjects; however, none of the saliva specimens contained detectable IgM. The possible origin of the IgA antibodies to VCA in the saliva of NPC patients is discussed. (7 refs.)

**77-2793 Immunolectron Microscopic Studies of Antibodies in Mouse Sera Directed Against Mouse Mammary Tumor Virus.** (Eng.) Miller, M. F. (Experimental Biology Div., Abbott Labs., N. Chicago, IL 60064) Dmochowski, L.; Bowen, J. M. *Cancer Res* 37(7): 2086-2091; 1977.

Indirect immunoferritin and fixed immunofluorescence tests were carried out on (1) sera of mice hyperimmunized with isologous mouse mammary tumor virus (MMTV) particles or isologous MMTV-producing mammary tumor-bearing and tumor-free mice of several inbred strains. Sera were tested against MMTV produced by C3H/HEJ/Tex tissue culture cells (MMT-1). Mammary tumor-bearing A/Dm, C3H/HeTex, and RIII/Dm mice and apparently tumor-free A/Dm mice developed naturally occurring nonprotective anti-MMTV antibodies. Sera of tumor-free C3H/HeTex, RIII/Dm, and C57BL/6/Tex female mice and A/Dm, C3H/HeTex, and RIII/Dm male mice did not contain anti-MMTV antibodies. Indirect immunoferritin and fixed immunofluorescence labeling of MMTV particles was prevented by absorbing the sera with purified MMTV particles. The results demonstrate the relationship of naturally occurring anti-

MMTV antibodies in mouse sera to the presence of mammary tumors, confirm previous reports that mice are not tolerant to MMTV, and further establish the usefulness of the indirect immunoferritin procedure in studies of the immune response of mice. (28 refs.)

**77-2794 Indirect Immunofluorescence Detection of Human IgM and IgG Antibodies Against Herpes Simplex Virus Type 1 Induced Cell Surface Antigens.** (Eng.) Marttila, R. J. (Dept. Virology, Univ. Turku, 20520 Turku 52, Finland) Kalimo, K. O. *Acta Pathol Microbiol Scand [B]* 85(3): 195-200; 1977.

Infected HeLa coverslip cultures were used to demonstrate the presence of human IgM and IgG in nine patients with herpes simplex virus type 1 primary infections. All five patients with recurrent herpes infections demonstrated only IgG antibodies. These titers paralleled the radioimmunoassay antibody titers closely but at a reduced level. (23 refs.)

**77-2795 Blocking Effect of Rheumatoid Factor on the In Vitro Cytotoxicity of Lymphoid Cells from Carcinoma Patients.** (Eng.) Saksela, E. (Third Dept. Pathology, Univ. Helsinki, 00290 Helsinki 29, Finland) Pyrohonen, S.; Timonen, T.; Teppo, A. M.; Wager, O.; Penttinen, K. *Scand J Immunol* 5(9): 1075-1080; 1976.

The blocking effect of rheumatoid factor (RF) on the in vitro cytotoxicity of lymphoid cells from patients with ovarian tumors was studied. These tumor cells and those from an established cell line from a transitional cell carcinoma of the human urinary bladder (RT-4) were exposed to (RF)-active cryo-immunoglobulin M (IgM) preparations from two patients with IgG-IgM cryoglobulinemia and to mononuclear cells from patients with either ovarian or bladder tumors. When precultured with effector cells, these RF preparations blocked the tumor-specific in vitro toxicity of the mononuclear cells as determined by microcytotoxicity assay. These results indicate that human RF factor can interfere with the tumor-specific cytotoxicity of ovarian or bladder carcinoma patients' peripheral blood lymphoid cells in vitro. Preincubation of the target cells with RF had no effect, indicating that the blocking was mediated through the effector cells; the blocking effect was due to the IgM RF. (19 refs.)

**77-2796 The Presence of Immunoglobulins and Receptors for Sheep Red Blood Cells in Human Lymphoblastoid Cell Lines Simultaneously Carrying the Epstein-Barr Virus and Cytomegalovirus Genomes.** (Fre.) Bourkas, A. E. (Department de Microbiologie, Universite de Montreal, Hopital Sainte-Justine, Montreal, Quebec, H3T 1C5, Canada) Okada, W.; Menezes, J.; Joncas, J. H. *Ann Immunol (Paris)* 128C (1/2): 215-217; 1977.



The lymphoblastoid cell lines EB-SD-2, EB-M-1 and T-D-1, which carry both the Epstein-Barr and cytomegalovirus genomes, were characterized. The first two were found to be derived from B lymphocytes; the third was composed of a mixed cell population, the majority B type cells. (12 refs.)

- 77-2797 **Lysis of Human Cultured Lymphoblastoid Cells by Cell-induced Activation of the Properdin Pathway.** (Eng.) Theofilopoulos, A. N. (Dept. Cellular and Developmental Immunology, Scripps Clinic and Res. Foundation, La Jolla, CA 92037) Perrin, L. H. *Science* 195(4281): 878-880; 1977.

Human cultured lymphoblastoid cells bearing C3b immune adherence receptors were found to activate the properdin pathway, bind components of the pathway, and undergo lysis. This mechanism may represent the in vivo surveillance to limit the growth of B-type lymphomas and lymphoblastic leukemia cells. (23 refs.)

- 77-2798 **Enzymatic Analysis of the Insulin Receptor on Activated T Cells (Meeting Abstract).** (Eng.) Reynolds, T. C. (Peter Bent Brigham Hosp., Boston, MA 02115) Helderman, J. H.; Carpenter, C. B.; Strom, T. B. *Fed Proc* 36(3): 1235; 1977. (no refs.)

- 77-2799 **The IB-Peptide Marker and the Ly-3 Surface Alloantigen: Structural Studies of a VK-Region Polymorphism and a T-Cell Marker Determined by Linked Genes.** (Eng.) Gottlieb, P. D. (Center Cancer Res., Massachusetts Inst. Technology, Cambridge, MA 02139) Durda, P. J. *Cold Spring Harbor Symp Quant Biol* 41: 805-815; 1977.

Autoradiography of peptide maps of light (L) chains from AKR/J and DBA/2J mice has revealed a IB-peptide marker in the V region. Further studies of amino acids found in each position of the cystine (Cys I) hexapeptides isolated from normal AKR/J L chains suggested that the marker is in  $\kappa$ -type L chains. This marker appears to be closely linked to the lysine (Ly-2 and Ly-3) loci that determine the expression of T-lymphocyte surface antigens, and several hypotheses that might account for this linkage are discussed. Mechanisms by which expression of the marker may be regulated are described. Isolate of IB-positive myeloma proteins should prove useful for investigating the extent of VK-region polymorphism governed by Ly2,3-linked genes. Studies using specific anti-Ly-3 sera have provided information on the structure of Ly-3 alloantigens. Exploration of the relationships among the linked loci is recommended to determine their possible involvement in immune phenomena. (37 refs.)

- 77-2800 **Lymphocyte Surface Markers in Malignant Lymphomas (Meeting Abstract).** (Eng.) Payne, S. V. (Univ. Dept. Pathology, South Lab. and Pathology Block, Southampton General Hosp., Tremona Road, Southampton SO9 4XY, England) Smith, J. L.; Wright, D. H. *Br J Cancer* 35(2): 251-252; 1977. (1 ref.)

- 77-2801 **Lymphoblastogenesis Inhibitory Factor Produced by Human Lung Cancer Cell Lines.** (Eng.) Han, T. (Medicine B Dept., Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263) Minowada, J. *J Surg Oncol* 9(3): 243-248; 1977.

A comparative study of the inhibitory effect of cell-free supernatants (CFS) from human lung cancer cell lines (ChaGo and PC-1) and from a human embryo fibroblast cell line (nonneoplastic) on lymphocyte blastogenesis indicated that both lung cancer cell lines produced a large amount of the inhibitory factor. Production of such a factor from the nonneoplastic cell line was minimal or absent. The inhibitory effect of the CFS of the lung cancer cell line was not due to direct cytotoxicity. It was partially sensitive to heat treatment. A significant inhibition was still seen when the CFS of the cancer cell lines were present for 1 hr prior to the addition of phytohemagglutinin. The possibility that the in vivo-production of the inhibitory factor contributes to the impaired cellular immunity of lung cancer patients is discussed. (16 refs.)

- 77-2802 **Lymphocyte: Tumour Cell Interaction in Oral Neoplasia (Meeting Abstract).** (Eng.) Noble, P. B. (Faculty Dentistry, McGill Univ., P.O. Box 6070, Station A, Montreal, Quebec, Canada H3C 3G1) Bentley, K. C. *Br J Cancer* 35(2): 252; 1977. (no refs.)

- 77-2803 **Cellular Basis of Persistent Lymphocytosis in Cattle Infected with Bovine Leukemia Virus.** (Eng.) Kenyon, S. J. (Section Viral Oncology, Comparative Leukemia Studies Unit, Sch. Veterinary Medicine, Univ. Pennsylvania, Kennett Square, PA 19348) Piper, C. E. *Infect Immun* 16(3): 891-897; 1977.

Peripheral blood lymphocytes (PBL) from 14 animals injected with bovine leukemia virus (BLV) and 14 BLV-free cattle were examined by the membrane immunofluorescent antibody technique to detect surface immunoglobulin (S-Ig) and by the erythrocyte-antibody-complement (EAC) rosette test for the detection of complement receptors. Direct comparisons of the percentages of S-Ig-bearing cells and EAC rosette-forming cells in both infected and BLV-free animals showed no evidence for the presence of a substantial population bearing one surface marker but not the other. Cells with surface markers characteristic of B lymphocytes were responsible for most of the increase in PBL that can accompany BLV infection. The release of infectious BLV and the spontaneous up-

take of thymidine by short-term cultured PBL from BLV-infected cattle were also studied. The results indicate that both of these activities are functions of B lymphocytes. (14 refs.)

77-2804 **Studies of the Classification and Function of Lymphocytes in Normal and Feline Leukemia Virus Infected Cats (Meeting Abstract).** (Eng.) Cockerell, G. L. (Ohio State Univ., Columbus, OH 43210) *Diss Abstr Int [B]* 37(8): 3696-3697; 1977. (no refs.)

77-2805 **Properties of Continuous T-Lymphocyte Cell Cultures of Marmoset Monkeys (Meeting Abstract).** (Eng.) Falk, L. (Dept. Microbiology, Rush-Presbyterian-St. Lukes Medical Center, Chicago, IL) Johnson, D.; McGrath, P.; Schudel, A.; Deinhardt, F. *Scand J Immunol* 6(6/7): 695-696; 1977. (no refs.)

77-2806 **Normal and Neoplastic Maturation of T-Lineage Lymphocytes.** (Eng.) Weissman, I. L. (Lab. Experimental Oncology, Dept. Pathology, Stanford Medical Sch., Stanford, CA 94305) Baird, S.; Gardner, R. L.; Papaioannou, V. E.; Raschke, W. *Cold Spring Harbor Symp Quant Biol* 41: 9-21; 1977.

Two techniques were used to analyze the hematolymphoid pathway leading to the appearance of T lymphocytes. Orthotopic synchronic transfer of yolk sac hematopoietic cells to the yolk sac cavity of 8-day-gestation mouse embryos demonstrated the eventual movement of this lineage to bone marrow. In a second series of experiments, > 90% of recent thymus-cell migrants appeared to be cortisone-sensitive in the periphery. Antigen-induced blastogenesis of thymus-cell migrants resulted in transformation to large pyroninophilic cells. Analysis of the binding of in vitro-produced, highly purified, labeled oncornaviruses on various lymphoma and leukemia cell lines demonstrated the presence of murine leukemia virus (MuLV)-specific binding sites on most T lymphomas induced by these viruses. Quantitative inhibition of C58NTD thymoma cells by cold MSV/MLV (Moloney sarcoma-leukemia virus complex) indicated that the binding sites are finite in number, specific, and do not represent virus-virus aggregation at the cell surface. The possible role of these binding sites in neoplastic transformation remains to be determined. (28 refs.)

77-2807 **Characteristics of the Immature Cells Involved in T Cell-mediated Enhancement of Syngeneic Tumor Growth.** (Eng.) Small, M. (Dept. Cell Biology, Weizmann Inst. Science, Rehovot, Israel) *J Immunol* 118(5): 1517-1523; 1977.

Nonsensitized thymocytes injected together with  $2.5 \times 10^4$  syngeneic Lewis lung carcinoma cells into inbred C57BL/6J male mice produced a tumor enhancement that was less pronounced than that obtained with tumor-sensitized thymocytes. Rapidly dividing, tumor-enhancing cells appeared to be eliminated from the thymus of donor mice at 1 day after im administration of 5 mg hydrocortisone acetate. However, at 48 hr after cortisone injection, the rapidly dividing precursor cells that repopulated the depleted thymus appeared to be capable of enhancing tumor growth. Therefore, these cells exhibited the characteristics of cells in the early stages of thymic processing. Treatment of the immature thymocytes with thymic humoral factor, which is a thymic hormone functioning in T-cell maturation, eliminated their capacity to enhance tumor growth. The results support the hypothesis that interaction between mature T cells and syngeneic tumors triggers tumor inhibition, whereas immature T cells in contact with a tumor stimulus enhance tumor growth. (30 refs.)

77-2808 **Cyclophosphamide-sensitive T Lymphocytes Suppress the In Vivo Generation of Antigen-specific Cytotoxic T Lymphocytes.** (Eng.) Rollinghoff, M. (Institut für Medizinische Mikrobiologie der Universität Mainz, Hochhaus Augustusplatz, 65 Mainz, Germany) Starzinski-Powitz, A.; Pfizenmaier, K.; Wagner, H. *J Exp Med* 145(2): 455-459; 1977.

Pretreatment of mice with a single dose of cyclophosphamide (100 mg/kg) converted their state of low immune responsiveness to a state of high responsiveness. This conversion can be reverted back to a state of low responsiveness by reconstituting the cyclophosphamide-treated mice with normal T cells. The cyclophosphamide-sensitive suppressor T cells probably block the final antigen-independent phase of the differentiation pathway of cytotoxic T lymphocytes. (13 refs.)

77-2809 **Concanavalin A-Mediated Activation of Antigen-primed Lymphocytes into Secondary Cytotoxic Lymphocytes.** (Eng.) Bonavida, B. (Dept. Microbiology and Immunology, Sch. Medicine, Univ. California Los Angeles, Los Angeles, CA 90024) *J Exp Med* 145(2): 293-301; 1977.

Evidence is presented that splenocytes primed to an allograft can be activated by concanavalin A (Con A) into secondary cytotoxic T lymphocytes (CTL) with specificity for the primary alloantigens. The cell-mediated cytotoxicity was found to be independent of Con A and was not affected by the Con A-specific inhibitor,  $\alpha$ -methyl-D-mannose pyranoside. It was suggested that activation of prekiller cells by Con A into CTL may be mediated by way of the same or similar receptors normally triggered by the stimulating antigens. (17 refs.)



**77-2810 T and B Cells and Delayed-type Skin Reactions in Asbestos Workers (Meeting Abstract). (Eng.)**

Lange, A. (Dept. Occupational Diseases, Medical Sch., Wrocław, Poland) Skibinski, G. *Scand J Immunol* 6(6/7): 720; 1977. (no refs.)

**77-2811 Fusion of T and B Cells. (Eng.)** Kohler, G. (MRC Lab. Molecular Biology, Cambridge, England CB2 2QH) Pearson, T.; Milstein, C. *Somatic Cell Genet* 3(3): 308-312; 1977.

Hybrid cells were prepared by fusing an immunoglobulin (Ig)-secreting mouse myeloma line (B cell) with an allogeneic T-cell lymphoma that expresses the surface antigen Thy 1. The resulting hybrids expressed H2 antigens of both parental cells and secreted the Ig of the myeloma parent, but they did not express the Thy 1 antigen of the lymphoma parent. Twenty-one hybrids were formed from fusion of the same myeloma line with trinitrophenylated-sheep RBC-primed spleen cells. Most of the hybrid lines exhibited the characteristics expected for the fusion of the myeloma to B lymphocytes. No hybrids between the myeloma line and spleen T cells were identified, since none of the hybrids expressed the T-cell-specific antigen Thy 1. Possible reasons for the failure to obtain hybrids with T-cell characteristics are discussed. (no refs.)

**77-2812 Critical Study of the Mononuclear Leukocyte Morphology Based on Scanning Electron Microscopy in Normal Subjects and in Patients with Lymphoid or Monocytoid Proliferative Disorders. Comparison with the T, B or Null Cell Membrane Phenotypes. (Eng.)** Dantchev, D. (Institut de Cancerologie et d'Immunogenetique, Hopital Paul Brousse, Service d'Hematologie de l'Institute Gustav Roussy, 14-16 bis, ave. Paul Vaillant-Coutureir, 94800, Villejuif, France) Belpomme, D. *Biomedicine* 27: 202-222; 1977.

Mononuclear WBC from 104 patients with chronic or acute lymphoid and monocytoid proliferative disorders, 1 patient with toxoplasmosis, 2 patients with mononucleosis, and from 12 normal subjects were studied by scanning electron microscopy. Correlations between the morphologic findings and immunologic T- and B-lymphocyte properties were tested in a double-blind study. Six main types of cell surfaces were distinguished in both pathological and normal WBC. Type 1 was characterized by completely smooth surfaces, type 2 by smooth undulated surfaces, type 3 by a partially villous surface, type 4 by a completely villous surface, type 5 by numerous surface blebs, and type 6 by completely ruffled surfaces. The majority of the T lymphocytes in normal subjects were villous and indistinguishable from B lymphocytes, but a fair correlation between T- and B-membrane phenotype and cell surface appearance was observed in the leukemia and lymphoma patients. In B-cell disorders, cell surfaces were most often types 3 or 4, sometimes type 5; in T-cell disorders, the

surfaces were either types 1 or 2. Cell surfaces in all monocytoid disorders were classified as type 6. In certain cases of lymphosarcoma, hairy cell leukemia, and Hodgkin's disease, cell surface properties could not be correlated with immunologic findings. This may be due either to technical problems or to cell membrane modifications related to functional or cell cycle-dependent variations. It is concluded that scanning electron microscopy, combined with other studies, can be helpful in classifying the leukemias and hematosarcomas (54 refs.)

**77-2813 Mechanism of P-815 Mastocytoma-mediated Suppression of Lymphocyte Reactivity (Meeting Abstract). (Eng.)** DeLustro, F. A. (State Univ. New York, Upstate Medical Center, Syracuse, NY 13210) *Dis. Abstr. Int. [B]* 37(8): 3861; 1977. (no refs.)

**77-2814 Reversible Inhibition of Human Peripheral Lymphocyte DNA Synthesis by an Extract of Breast Cancer Cell Line SKBR-3. (Eng.)** Lundy, E. G. (Dept. Surgery, Univ. Miami Sch. Medicine, Miami, FL 33152) Sorokin, C. F.; Meltz, S. K.; Glade, P. R. *J. Surg. Res.* 22(6): 654-659; 1977.

An extract derived from a malignant breast cell line (SKBR-3) was tested for its ability to inhibit mitogen- and antigen-stimulated human peripheral lymphocytes. The extract consistently inhibited DNA synthesis in phytohemagglutinin-stimulated lymphocytes by 32% to 72%, and the inhibition was most pronounced at an effector/target (E/T) ratio of 3:1. Essentially complete reversibility (87%-102%) of the inhibition was demonstrated. At E/T ratios of 12:2, the SKBR-3 extract significantly inhibited DNA synthesis in SKBR-3 cells. Inhibition of antigen-stimulated lymphocytes could not be assessed, as the SKBR-3 monolayer cultures were not stimulatory to peripheral lymphocytes. The inhibitor is trypsin-sensitive, thermolabile, and DNase-resistant. The role of endogenous lymphocyte inhibitors in depressing responses in cancer patients is discussed briefly. (20 refs.)

**77-2815 Human Lymphocyte-Mouse Myeloma Somatic Cell Hybrids: Selective Hybrid Formation. (Eng.)** Schwaber, J. (Immunology Div., Children's Hosp. Medical Center, Boston, MA 02115) *Somatic Cell Genet* 3(3): 295-302; 1977.

Fusion of unfractionated human lymphocytes with mouse myeloma cells resulted in proliferating hybrid colonies, almost all producing human immunoglobulin (Ig). The possibility that this was the result of selective hybrid formation by B lymphocytes, rather than the induction of Ig genes in T lymphocytes, was examined. Unfractionated peripheral lymphocytes and B lymphocytes from patients with the common variable form of agammaglobulinemia formed prolife-

rating somatic cell hybrid colonies. In contrast, peripheral lymphocytes from an agammaglobulinemia patient who lacked B lymphocytes failed to produce somatic cell hybrids with three different myeloma parent cell lines. Fractionation of hybrid-forming lymphocytes on discontinuous albumin gradients confirmed the finding that only B lymphocytes formed hybrid colonies. B, T, and precursor lymphocytes all had Sendai virus receptors, as evidenced by viral agglutination. The results demonstrate that fusion of human lymphocytes with mouse myeloma cells results in selective hybrid formation, not activation of Ig genes in disparate cell types. However, only B-lymphocyte-mouse melanoma heterokaryons form hybrid cells. (24 refs.)

77-2816 **Immunological Properties of Anti-tumor Immune RNA (Meeting Abstract).** (Eng.) Evans, S. B. (Ohio State Univ., Columbus, OH 43210) *Diss Abstr Int [B]* 37(8): 3778; 1977. (no refs.)

77-2817 **I-RNA: Synthesis and Mechanisms of Action.** (Eng.) Jachertz, D. *In: Immune RNA in Neoplasia*. Fink, M. A., ed. (New York: Academic Press, Inc.) pp. 95-111; 1976.

Antigen-recognizing cells appear to synthesize an informational RNA (iRNA) that transmits information to antibody-synthesizing cells. Synthesis of iRNA in cellular and cell-free systems is described. In a cell-free system, the synthesized iRNA is not contaminated with other types of RNA's. Electron micrographs of the three types of iRNA separated by Cs<sub>2</sub>SO<sub>4</sub> gradient centrifugation, single-stranded, replicative intermediate, and double-stranded iRNA, are illustrated. The actions of iRNA in cell-free and cellular systems are summarized. iRNA not only instructs lymphoid cells to synthesize antibody but also the cell's own iRNA, by means of a regulator protein. In DBA/2 mice implanted with P815 mastocytoma and in Lewis rats injected with polyomavirus-transformed tumor cells, treatment with iRNA (10<sup>6</sup> molecules of iRNA-P815) inhibited tumor growth. However, cer-

tain variations in the amount of transplanted cells, the concentration of iRNA, and the time of treatment led to tumor enhancement. The possibility of using iRNA as a vaccine is discussed. (24 refs.)

77-2818 **Informational RNA Directed Synthesis of Virus Neutralizing Proteins in Cell-free Extracts Prepared from Rat Spleen and Mouse L Cells.** (Eng.) Koch, G.; Bilello, P.; Fishman, M.; Mittelstaedt, R.; Borriess, E. *In: Immune RNA in Neoplasia*. Fink, M. A., ed. (New York: Academic Press, Inc.) pp. 113-120; 1976.

Informational RNA (iRNA) within poly(A)-containing RNA isolated from either (1) MS2 phage- and poliovirus-exposed mouse peritoneal exudate (PE) cells or (2) T<sub>2</sub> phage-exposed rabbit PE cells was used to direct the synthesis of proteins in cell-free extracts prepared from rat spleen and mouse L cells, respectively. The properties of the in vitro-synthesized proteins were subsequently analyzed by the use of affinity chromatography on virus-agarose columns, virus-neutralizing assays, allotype characterization, and determination of monomeric and polymeric size. In both rat spleen and mouse L cell extracts, iRNA induced the synthesis of virus-specific neutralizing proteins that resemble antibodies. Virus-neutralizing activity was found in 19S proteins only, and it was amplified specifically by antiallotype serum. The results indicate that iRNA functions as messenger RNA and that it contains the coding capability to direct the synthesis of complete IgM antibodies. (13 refs.)

\* (Rev): 77-2437, 77-2438, 77-2439, 77-2440, 77-2441, 77-2442, 77-2444.

\* (Chem): 77-2483, 77-2484, 77-2556.

\* (Viral): 77-2658, 77-2659, 77-2661, 77-2674, 77-2677, 77-2683, 77-2684, 77-2689, 77-2690, 77-2694, 77-2697, 77-2703.

\* (Path): 77-2878.



## PATHOGENESIS

- 77-2819 **Human Breast Carcinomas: Marker Chromosomes Involving 1q in Seven Cases.** (Eng.) Cruciger, Q. V. (Dept. Medicine, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) Pathak, S.; Cailleau, R. *Cytogenet Cell Genet* 17(4): 231-235; 1977.

The presence of chromosome anomalies in seven cell lines of human breast carcinoma was investigated by Giemsa (G) banding techniques. Six lines were from a pleural effusion and one was from a metastatic brain tumor. The modal chromosome number varied between 40 and 66, depending on the line. The distal segment of the long arm of chromosome No. 1 (1q) was involved in translocations in all seven lines. The translocated partner chromosome was less constant, being chromosome Nos. 3, 5, 7, 11, or 12, depending on the cell line observed. A translocation involving chromosome No. 1 seems to be a common denominator of breast carcinoma; with confirmation from further study, it could possibly be used as a marker for the disease. (10 refs.)

- 77-2820 **Cytospectrophotometric Study of the DNA Content in Dysplasia and Mammary Gland Carcinoma.** (Rus.) Zolotarevskii, V. B. (Dept. Pathological Anatomy, Central Scientific Res. Lab., I. M. Sechenov First Moscow Medical Inst., Moscow, USSR) Popov, M. S. *Arkhi Patol* 39(1): 25-31; 1977.

The DNA content of the epithelial cells of breast lesions (dysplasia, carcinoma) was measured cytospectrophotometrically. Normal lymphocytes were used as a reference for the DNA value corresponding to a diploid number of chromosomes. Specimens of surgically resected breast tissue were obtained from 22 patients aged 26-66 yr. Histologically, the lesions were intracanalicular fibroadenoma (2), duct papilloma (1), proliferative fibroadenomatosis (4), intraduct carcinoma in situ and early invasive duct carcinoma (6), adenocarcinoma (2), solid carcinoma (2), scirrhous carcinoma (2), mucinous carcinoma (2) and squamous cell carcinoma (1). The epithelial nuclei of the dysplastic tissue had DNA values equivalent to a triploid number of chromosomes [approx 3 nanograms (ng)] but in the invasive carcinomas the av DNA content of the nuclei was significantly higher ( $> 4$  ng). The significance of these findings is discussed with reference to the possible use of DNA content as a criterion of early malignant lesions. (19 refs.)

- 77-2821 **Pseudoadenoid Cystic Carcinoma of the Breast.** (Eng.) Harris, M. (Dept. Pathology, Withington Hosp., Manchester, England) *Arch Pathol Lab Med* 101(6): 307-309; 1977.

An example of a cribriform intraductal carcinoma that closely resembled an adenoid cystic carcinoma was reported in a 46-yr-old woman. In November 1973 she complained of milky discharge from both nipples and, on examination, was thought to have bilateral fibroadenosis. In February 1975, the patient complained of a small lump in the upper outer quadrant of the right breast. An excision biopsy of the lump was performed, and it was followed shortly by a simple mastectomy with biopsy of two axillary lymph nodes. The tumor consisted of well-defined, rounded or lobulated, variably sized groups of small dark epithelial cells that enclosed large and small cribriform spaces containing plugs of mucinlike material. The tumor cell groups were bounded by a thin, intact, basement membrane inside which was a peripheral, discontinuous layer of myoepithelial cells. The diagnosis of the tumor could not be made safely by routine histologic techniques alone. The true nature of the tumor was revealed by electron microscopy. Ultrastructural examination is a useful alternative tool in the differentiation of adenoid carcinoma of the breast from cribriform intraductal carcinoma. (13 refs.)

- 77-2822 **Adenoid Cystic Carcinoma of the Breast. Light and Electron Microscopy and a Brief Review of the Literature.** (Eng.) Qizilbash, A. H. (Dept. Lab. Medicine, Henderson General Hosp., 711 Concession St., Hamilton, Ontario, Canada L8V 1C3) Patterson, M. C.; Oliveira, K. F. *Arch Pathol Lab Med* 101(6): 302-306; 1977.

The cases of two patients with adenoid cystic carcinoma of the breast are presented. A 28-yr-old woman was admitted to the hospital in January 1966 with a complaint of bloody discharge from the nipple of the left breast of 4 wk duration. A clinical diagnosis of subareolar papillomatosis was made, and the patient underwent surgery. The patient was seen again in June 1966, when a 2.0- × 2.0- × 3.0-cm mass was palpated at the site of the previous surgery. A frozen-section diagnosis of recurrent cylindromatous adenocarcinoma was made, and a modified radical mastectomy was performed. The patient has been alive and symptom-free for the past 9.5 yr. A 43-yr-old woman was admitted to the hospital with a 10-yr history of a lump in the right breast that recently had increased in size. A diagnosis of adenoid cystic carcinoma

was made, and a modified radical mastectomy was performed in July 1975. There have been about 95 reports of this type of lesion. Electron microscope investigations confirm that many of the cystic spaces observed by light microscopy are extracellular compartments enclosed by tumor cells. Many of the tumor cells contain densely packed fibrils, supporting the myoepithelial origin of the tumors. (43 refs.)

- 77-2823 **Carcinoma of the Breast with Multinucleated Reactive Stromal Giant Cells. A Light and Electron Microscopic Study of Two Cases.** (Eng.) Factor, S. M. (Dept. Pathology, Albert Einstein Coll. Medicine, 1300 Morris Park Ave. Bronx, NY 10461) Biempica, L.; Ratner, I.; Ahuja, K. K.; Biempica, S. *Virchows Arch [Pathol Anat]* 374(1): 1-12; 1977.

Two unusual carcinomas of the breast are described. Each contained nests of infiltrating neoplasm situated within stromal lacunar spaces, and surrounded by numerous benign-appearing multinucleated giant cells. Within the stroma, there was extensive hemorrhage, hemosiderin pigment deposition, and large numbers of mononucleated inflammatory cells. The morphology of both tumors resembled the giant cell tumor of bone. Although a similar giant cell reaction has recently been described in association with a uterine leiomyosarcoma, there have been only two other examples of this entity in the breast, both reported > 40 yr ago in the French literature. This is the first report in which electron microscopy confirmed the benign histiocytic nature of the giant cells. These cells had many of the ultrastructural features of multinucleated giant cells described in tissue culture, skeletal osteoclastomas, and foreign-body granulomas. It is proposed that the giant cells arise from fusion of mononucleated stromal cells and most likely are reactive histiocytic elements related in some way to the tumor cell nests. Further studies of these unusual neoplasms are needed to determine if the giant cell reaction affects prognosis. (22 refs.)

- 77-2824 **Axillary Micrometastasis and Macrometastasis in Carcinoma of the Breast.** (Eng.) Attiye, F. (Dept. Surgery and Pathology, Memorial Sloan-Kettering Cancer Center, New York, NY) Jensen, M.; Huvos, A. G.; Fracchia, A. *Surg Gynecol Obstet* 144(6): 839-842; 1977.

The results are reported of a 14-yr follow-up study of 105 patients (mean age at diagnosis, 51.5 yr) who underwent radical mastectomy for breast carcinoma metastatic to the axillary nodes. Patients with micrometastasis alone have a good prognosis with 10- and 14-yr survival rates of 85% and 77%, respectively, if only level I is involved. No patient had micrometastasis in more than three nodes. Patients with macrometastasis at any level or with four or more positive nodes have a poor prognosis. (6 refs.)

- 77-2825 **The Correlation Between the Latent Period Duration of Induced Neoplastic Process in the Mammary Glands, Season of the Year, and Thyrotropic Function of the Hypophysis (Meeting Abstract).** (Rus.) Sukacheva, O. A. (Kharkov Scientific Res. Inst. Endocrinology and Hormone Chemistry, Kharkov, USSR) *Vopr Onkol* 22(12): 80; 1976. (no refs.)

- 77-2826 **Iron and Ferritin Studies in Mouse and Human Mammary Glands and Tumors (Meeting Abstract).** (Eng.) Seman, G. (Dept. Molecular Carcinogenesis, Univ. Texas System Cancer Center M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) Gallager, H. S. *Proc Am Assoc Cancer Res* 18: 57; 1977. (no refs.)

- 77-2827 **Endometrioid Carcinoma of the Ovary: Clinical, Histologic, and Ultrastructural Observations (Meeting Abstract).** (Eng.) Kanbour, A. I. (Dept. Pathology, Univ. Pittsburgh Medical Sch., Pittsburgh, PA 15213) Salazar, H. *Lab Invest* 36(3): 342; 1977. (no refs.)

- 77-2828 **Squamous Carcinoma Arising in Benign Cystic Teratoma of the Ovary. A report of Four Cases and Review of the Literature.** (Eng.) Krumerman, M. S. (Dept. Pathology, Roosevelt Hosp. and Coll. Physicians and Surgeons Columbia Univ., New York, NY) Chung, A. *Cancer* 39(3): 1237-1242; 1977.

Four cases are reported in which squamous carcinoma of the ovary developed from benign ovarian cystic teratomas. The literature showed that this malignant transformation occurs in 1-30% of cases. Histopathology of the tumors is described. (15 refs.)

- 77-2829 **Gene Mapping Using Ovarian Teratomas (Meeting Abstract).** (Eng.) Povey, S. (MCR Human Biochemical Genetics Unit, Galton Lab., Univ. Coll., London, England) *Heredity (London)* 38(pt 2): 273-274; 1977. (no refs.)

- 77-2830 **Mechanism of Development and Morphology of Secondary Carcinomas of the Oviducts in Primary Uterine Corpus Carcinoma.** (Eng.) Anbrokh, G. B. (Kurgan Municipal Hosp., Kurgan, USSR) Anbrokh, Ya. M. *Neoplasma* 23(5): 549-557; 1977.

The incidence and morphology of metastases in 284 fallopian tubes from 148 patients with primary cancer of the uterine corpus were investigated. Metastases were found in 23 oviducts of 17 patients, in 10 of whom only the fallopian tubes



were affected. The metastases were of lymphogenic (12 oviducts), implantation (7), or lymphogenic-implantation (4) origin. They were localized mainly in the ampullary regions of the oviducts. Macroscopic changes in the form of thickening and condensation or in the form of tumor nodules were noted in seven oviducts. The lymphogenic metastases were observed more often in the subserosal and muscular layers than in the mucosal layers; however, all layers of the oviduct wall were occasionally involved. Implantation metastases in the form of micronodules or multicellular agglomerates were detected in the serosa more often than in the mucosa. Differences in the histologic structure and growth patterns of secondary and primary oviduct carcinomas are discussed together with the role of the fallopian tubes in the dissemination of metastases from primary uterine corpus carcinoma. (21 refs.)

- 77-2831 **Ultrastructure of Mullerian Adenosarcoma of the Uterus (Meeting Abstract).** (Eng.) Feldman, P. S. (Dept. Pathology, Univ. California, San Diego, La Jolla, CA 92093) Katzenstein, A. L.; Askin, F. B. *Lab Invest* 36(3): 338; 1977. (no refs.)

- 77-2832 **Follow-up Studies of Cytologically Detected Precancerous Lesions (Dysplasia) of the Uterine Cervix.** (Eng.) Nasiell, K.; Nasiell, M.; Vaclavinkova, V.; Roger, V.; Hjerpe, A. In: *Health Control in Detection of Cancer. Proceedings of a Symposium on Health Control in Detection of Cancer held by the Skandia Group, 23-25 September, 1975. The Skandia Group. (Stockholm, Sweden):* pp. 244-256; 1976.

The relationship between the development of dysplastic lesions and their progression toward or regression from cervical carcinoma is analyzed. Of the 1,700 women who participated in the study, 555 had mild dysplasia, 892 had moderate dysplasia, and 265 had severe dysplasia. The patients were examined and assayed via vaginal smear and colposcopy, and each group was subdivided into those who had been subjected to punch biopsy and those who had not. Emphasis in this study was placed on the group of patients with moderately dysplastic tumors, and it was found that 50% of the patients with lesions showed regression, 21% showed persistence of the lesion, and 25% showed progression of the lesion to severe dysplasia. It was suggested that, for patients in this category, radical therapy is not indicated and that a control period of about 1 yr is justified before conization is considered. The trauma of punch biopsy was not found to have any effect on the development of the lesions. (27 refs.)

- 77-2833 **An Immunofluorescent Study of Basement Membranes in Squamous Cell Carcinoma of the Cervix, Vagina and Vulva.** (Eng.) Pertschuk, L. P. (Dept. Pathology, Box 25, Downstate Medical Center, SUNY, 450

Clarkson Ave., Brooklyn, NY 11203) Boyce, J. G.; Urcuyo R. *Obstet Gynecol* 49(4): 417-420; 1977.

Immunofluorescent techniques were employed in the study of basement membranes of 33 samples of in situ or invasive squamous cell carcinomas of the cervix uteri, vagina or vulva. Antibody to basement membrane, obtained from patients with bullous pemphigoid, possessed a high titer as determined by indirect immunofluorescence examination on substrates of monkey esophagus. Clearly defined basement membranes were seen in all nine cases of in situ carcinoma from the cervix and vagina. Among the 24 specimens from patients with squamous cell carcinoma, moderately well-formed basement membranes were seen in 13 of 16 from the cervix, in both specimens from the vagina and in 3/6 samples from the vulva. These findings support the premise that penetration of the basement membrane is not a valid criterion for distinguishing in situ from invasive squamous cell carcinoma. (22 refs.)

- 77-2834 **Condyloma Acuminatum and Squamous Carcinoma of the Vulva.** (Eng.) Rhatigan, R. M. (Dept. Pathology, 655 W. Eighth St., Jacksonville, FL 32209) Saffos, R. O. *South Med J* 70(5): 591-594; 1977.

Clinical and histopathologic data are reported for three patients in whom condyloma acuminatum and squamous cell carcinoma occurred in the vulva and for a fourth patient in whom the condyloma was followed by squamous dysplasia. This association has been reported previously in both male and female genitalia and in the perianal area. In the four cases, cancer or squamous dysplasia followed long-standing or extensive condyloma, but there was no evidence of transformation from condyloma to carcinoma. In one of the cases, areas of squamous dysplasia occurred in multiple foci within a large condyloma. It is not known whether condyloma acuminatum is a precancerous skin lesion, but long-standing or extensive condylomas should alert the clinician to the possibility of carcinoma. (20 refs.)

- 77-2835 **Condylomatous Lesions of Cervix and Vagina: Cytologic Patterns (Meeting Abstract).** (Eng.) Meisels, A. (Cytopathology Lab., St-Sacrement Hosp. Quebec, P.Q. Canada) *Acta Cytol (Baltimore)* 20(6): 582; 1976. (no refs.)

- 77-2836 **Clear Cell Adenocarcinoma of the Vagina and DES: A Scanning Microscopy, Transmission Electron Microscopy and Cytopathologic Study (Meeting Abstract).** (Eng.) Blair, O. M. (Dept. Pathology, Saint Louis Univ. Sch. Medicine, St. Luis, MO) Cavanagh, D.; Hovadhanakul, P.; Taylor, H. B. *Acta Cytol (Baltimore)* 20(6): 587-588; 1976. (no refs.)

77-2837 **Microcalcifications in Testicular Germ Cell Tumors. Orienting Study Concerning Their Diagnostic Utilization.** (Ger.) Wurster, K. (Pathologisches Institut der Universität Heidelberg, Im Neuenheimer Feld 220/21, D-6900 Heidelberg, W. Germany) Menges, V. *Virchows Arch [Pathol Anat]* 374(1): 45-62; 1977.

Microscopic and radiographic evaluation of the histologic slides and paraffin-embedded tissue specimens of 129 germ cell tumors showed that 46.4% of the testes with seminoma and 68.3% of the testes with teratoma display microcalcifications. They appear as round or roundish psammomatous bodies, irregularly shaped dystrophic calcifications, or in teratomas, as particles of bone tissue or calcified cartilage. The psammomatous bodies are located within tubules in compressed residual testicular tissue arranged in a shell-like zone around the tumor mass, but the dystrophic calcifications and bone and cartilage tissues are identified inside the tumor. Often > 10 microcalcifications/cm<sup>2</sup> are present. The diagnostic importance of these findings for clinical use is discussed. The diagnosis must be based on a radiographic method that will not be dangerous for testicular tissue. (39 refs.)

77-2838 **Forms of Human Prostatic Dysplasias (Meeting Abstract).** (Ger.) Kastendieck, H. (Hamburg, W. Germany) Altenahr, E. *Zentralbl Allg Pathol* 121(3): 300; 1977. (no refs.)

77-2839 **Metastatic Prostate Carcinoma to the Mandible: Report of Case.** (Eng.) Mesa, M. L. (100 Bergen St., Newark, NJ 07103) *J Oral Surg* 35(2): 133-135; 1977.

A case of metastatic prostate carcinoma to the mandible in a 52-yr-old man who was admitted with a well-developed firm nodule in the prostate, evidence of a possible cerebrovascular accident, and severe renal failure is described. Six months later, extensive skeletal involvement in the form of multiple radiodense lesions was found. The relatively infrequent occurrence of metastases in the jaw is due to the low content of hematopoietic marrow and decreased vascularization. (14 refs.)

77-2840 **Secondary Tumors of the Ureter Stump (Meeting Abstract).** (Dut.) Cuypers, L. H. (Sittard, Netherlands) *Ned Tijdschr Geneesk* 121(11): 470-471; 1977. (no refs.)

77-2841 **Membrane Changes of Invasive Human Transitional Carcinoma Cells (Meeting Abstract).** (Eng.) Hicks, R. M. (Dept. Histopathology, Middlesex Hosp.

Medical Sch., Riding House Street, London W1P 7LD, England) Chowaniec, J.; Newman, J. *Br J Cancer* 35(2): 250; 1977. (no refs.)

77-2842 **Ultrastructural Changes at the Epithelial-Mesenchymal Interface in Experimentally Induced Bladder Tumours (Meeting Abstract).** (Eng.) Chowaniec, J. (Dept. Histopathology, Middlesex Hosp. Medical Sch., Riding House Street, London W1P 7LD, England) Hicks, R. M. *Br J Cancer* 35(2): 254; 1977. (no refs.)

77-2843 **Bladder Cancer as a Disease of Hormone Imbalance in Nb Rats (Meeting Abstract)?** (Eng.) Noble, R. L. (Cancer Res. Center, Univ. British Columbia, Vancouver, Canada V6T 1W5) *Proc Am Assoc Cancer Res* 18: 131; 1977. (no refs.)

77-2844 **Bladder Tumor Associated with Phenacetin Abuse.** (Eng.) Tosi, S. E. (2 Maynard St., Hanover, NH 03755) Morin, L. J. *Urology* 9(1): 59-60; 1977.

A 56-yr-old man with a 20-yr history of ingestion of five or six phenacetin-containing analgesic tablets daily for knee pain developed 1.5-cm sessile papillary tumor on the right low posterior bladder wall. Histologic examination revealed a papillary transitional cell carcinoma of the bladder, low grade. Due to a possible correlation between phenacetin ingestion and transitional cell carcinoma of the renal pelvis and bladder the detrimental effects of phenacetin far outweigh its potential benefits. (8 refs.)

77-2845 **Urinary Amino Acid Excretion. Comparison of Normal Individuals and Patients with Bladder Cancer.** (Eng.) McGregor, R. F. (Dept. Lab. Medicine, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) Johnson, D. E.; Sharon, M. S.; Crawford, R.; Brown, B.; Johnston, D. *Urology* 9(5): 538-542; 1977.

Urinary amino acid excretion was studied by gas chromatography in normal individuals and in 118 patients with bladder carcinoma to determine whether differences existed between the two populations and if any alterations were related to the stage of the disease. A striking difference was noted in isoleucine excretion. It was not detectable in normal subjects, but a mean of 0.03 (patients with a previous history of bladder cancer) to 0.26 (Stage D4 patients) millimole/ml urine was found in the cancer patients. The monitoring of isoleucine content in the urine may be useful in distinguishing bladder cancer patients from normal subjects. Linear discriminant analysis suggested that excretion values for glycine, leucine,



proline, and glutamic acid, as well as cysteine concentrations, were altered in male patients according to the invasiveness of the disease. In women, valine, serine, aspartic acid, phenylalanine, and lysine concentrations varied according to the invasiveness of the disease. These findings suggest that measurements of urinary amino acids may be useful for the detection and staging of bladder carcinoma. (22 refs.)

- 77-2846 **Problems of Histogenesis and Classification of Nephroblastomas (Wilms' Tumor) in Children.** (Rus.) Sukhova, V. N. (Dept. Pathological Anatomy, Central Postgraduate Medical Inst., Moscow, USSR) *Arkhh Patol* 39(1): 32-38; 1977.

A histological classification of Wilms' tumor (nephroblastoma) based upon the degree of tumor differentiation was devised. Of 148 children with nephroblastoma, 42 had poorly differentiated tumors that were composed almost entirely of undifferentiated cells (Type I). Type II tumors (46 cases) included those in which the degree of tissue differentiation was more pronounced. Type III tumors (60 cases) was a composite group of different histologic types: nephrogenic tumors (16), mesenchymal tumors (13), myogenic tumors (8), and mixed tumors (23). It was concluded that the criteria for nephroblastoma are the presence of undifferentiated tissue (tumorous metanephrogenic blastoma) or tumorous nephrogenic structures at different stages of development. (45 refs.)

- 77-2847 **Study of the In Vitro Differentiation of Nephroblastoma (Wilms' Tumor) (Meeting Abstract).** (Fre.) Rousseau-Merck, M. F. (INSERM U-77, Hopital Necker, 149, rue de Sevres, 75730 Paris Cedex 15, France) Lombard, M. N.; Nezelof, C.; Mouly, H. *J Microsc (Paris)* 27(1): 22a; 1976. (no refs.)

- 77-2848 **Observation of Nervous Tissue in a Wilms' Tumor: Its Histogenetic Significance.** (Eng.) Kuo, T. T. (Dept. Pathology, Univ. Texas Medical Branch, Galveston, TX 77550) *Cancer* 39(3): 1105-1108; 1977.

A Wilms' tumor in a horseshoe kidney resected from a 13-mo-old girl contained nervous tissue with ganglion cells. The finding of neuroepithelial tissue in this tumor necessitates a reevaluation of tumor origin. (11 refs.)

- 77-2849 **Pulmonary Lymphangitic Carcinomatosis in Renal Adenocarcinoma.** (Eng.) Nunez, D. (Dept. Radiology, Hosp. St. Raphael, New Haven, CT 06511) Gonzalez-Serva, L.; Galloway, S. J. *Br J Radiol* 50(590): 142-143; 1977.

The case history of a 39-yr-old man with renal carcinoma

with an unusual invasion of the perivascular and peribronchial lymphatics is presented. This is the first reported case of this type of metastatic involvement from a renal tumor. (3 refs.)

- 77-2850 **Bronchial Carcinoma Metastasising to the Ischio-rectal Fossa.** (Eng.) Levack, B. (Chapel Allerton Hosp., Leeds, England) McCollum, C. N. *Br Med J* 1(6069): 1137-1138; 1977.

A 72-yr-old man presented with a 2-wk history of perianal pain made more intense by the passage of stools. The patient was afebrile, and he had an ischio-rectal mass that was not fluctuant. Exploratory operation showed a woody hard tumor occupying the entire ischio-rectal fossa. Histological examination revealed fat and muscle infiltrated with poorly differentiated squamous carcinoma consistent with an origin in the bronchus. Chest x-ray and bronchoscopy showed the presence of a growth arising from the right bronchus, and biopsy revealed a squamous cell carcinoma. (3 refs.)

- 77-2851 **Adenocarcinoma of the Pancreas Associated with Neurofibromatosis.** (Eng.) Keller, R. T. (Dept. Medicine, Univ. North Dakota Sch. Medicine, Grand Forks, ND 58201) Logan, G. M. *Cancer* 39(3): 1264-1266; 1977.

The established association of neurofibromatosis and secondary malignancies was further demonstrated by the report of a case of a 27-yr-old woman with pancreatic adenocarcinoma and neurofibromatosis. Since pancreatic cancer rarely occurs before the age of 30 yr, it is suggested that neurofibromatosis may predispose a patient to the development of secondary malignancies. (8 refs.)

- 77-2852 **Pancreatic Secretion in Hamsters with Pancreatic Cancer.** (Eng.) Reber, H. A. (Surgical Service--112, Veterans Admin. Hosp., 4150 Clement St., San Francisco, CA 94121) Johnson, F. E.; Montgomery, C.; Carl, W. R. *Surgery* 82(1): 34-41; 1977.

Pancreatic secretory abnormalities develop in most persons with pancreatic cancer, and they have been attributed to ductal obstruction. The possibility that abnormal secretion results instead from carcinogen-induced changes in the secreting cells was investigated in 50 male Syrian Golden hamsters given weekly sc injections of diisopropylnitrosamine (250 mg/kg). Survivors and age-matched controls were studied after 3.5 to 6.5 mo of treatment. Pancreatic secretion was stimulated by secretin or cholecystikinin (2 U/kg iv, as a bolus). After each stimulus, four 15-min collections of pancreatic juice were analyzed for HCO<sub>3</sub><sup>-</sup> and Cl<sup>-</sup> or total protein, amylase, trypsin, and chymotrypsin. The organs were examined histologically. The incidence of pancreatic ductal

carcinoma was 30% at 5 mo, 56% at 5.5 mo, and 100% at 6.5 mo. The animals without cancer either had hyperplasia of the duct epithelium or were histologically normal. Protein secretion and the histologic appearance of the acinar tissue were normal in all groups. The tumors did not obstruct the major ducts. In all treated animals, the pancreatic secretory response to secretin was of low volume, low max  $[HCO_3^-]$  and  $HCO_3^-$  output, and low  $[Cl^- + HCO_3^-]$ ; these changes progressed with time. The secretory abnormalities antedated the appearance of the neoplasms and were not caused by obstruction. (18 refs.)

**77-2853 Cystadenoma of the Pancreas. An Ultrastructural Study.** (Eng.) Lo, J. W. (Pathology Dept., Mercy Hosp. and Medical Center, Chicago, IL 60616) Fung, C. H.; Yonan, T. N.; Martinez, N. *Cancer* 39(6): 2470-2474; 1977.

An ultrastructural study of the tissue from a pancreatic cystadenoma indicated that the tumor may have arisen from embryonic nests. The results suggested that a depression mechanism may have allowed the tumor to switch to the production of large amounts of glycogen granules. (10 refs.)

**77-2854 Risk Factors and Early Diagnosis in Pancreatic Carcinoma.** (Eng.) Morgan, J. E. (Dept. Surgery, New England Medical Center Hosp. and Tufts Univ. Sch. Medicine, Boston, MA) Martyak, S. N. *Am J Gastroenterol* 67(3): 257-260; 1977.

The case history of a 6-yr-old black woman with adenocarcinoma of the pancreas is presented. She had multiple high risk factors for the disease (race, gallstones, diabetes, cholecystectomy) and died 6 wk after surgical treatment. A closer follow-up of the patients at risk is recommended. (15 refs.)

**77-2855 Liver Cirrhosis due to Breast Carcinoma Metastasis: So-Called Metastatic Carcinomatous Cirrhosis.** (Ger.) Breitfellner, G. (Institut für Pathologie, A-6807 Feldkirch, Austria) Dirschmid, K. *Schweiz Med Wochenschr* 107(7): 241-243; 1977.

Ten yr after undergoing a Rotter radical mastectomy for a carcinoma solidum simplex with lymph node metastases, as well as x-ray therapy before and after the operation, a woman, 44 yr old, presented with pains in the hip, enlarged liver and spleen, ascites icterus and other symptoms. Clinical and x-ray studies revealed retroperitoneal lymph node metastases and also metastases in the lungs, pleura, spine and liver. After improvement due to unspecified cytostatic therapy ceased, the ascites recurred and finally melena. Clinical and laboratory results indicated liver cirrhosis and portal hypertension and esophageal varices and liver metastases. There was no liver damage due to alcohol or hepatitis in the patient's histo-

ry. The patient died in a hepatic coma. Liver changes seen at autopsy were cirrhosis as a consequence of late metastases from a scirrhus mammary carcinoma. An unusual aspect of the case was the long symptom-free interval (9 yr), since the prognosis in such cases is poor. (6 refs.)

**77-2856 Hepatitis B Virus, Liver Cirrhosis and Cancer. An Electron Microscopic Study.** (Hun.) Kendrey, G. (Fovárosi László Korház, Korbontani és Korszovettani Osztály, Budapest, Hungary) *Magy Onkol* 20(4): 226-231; 1976.

The case history of a 61-yr-old man with chronic aggressive hepatitis and liver cirrhosis is presented. The hepatitis B virus core was found in the nucleus and the virus in the cytoplasm of many liver cells taken on biopsy. Postmortem examination 35 days after laparoscopy and 42 days after biopsy revealed primary liver carcinoma. The hepatitis B virus may have caused both the cirrhosis and cancer. (22 refs.)

**77-2857 Hepatitis B Virus and Primary Liver Carcinoma: Evidence for an Association Between Hepatitis B, Cirrhosis and Primary Liver Cancer.** (Eng.) Maupas, P. (Laboratoire de Virologie, Département de Microbiologie Faculté de Médecine, Tours, France) Coursaget, P.; Goudeau, A.; Drucker, J.; Sankale, M.; Linhard, J.; Diebolt, G. *Ann Microbiol (Paris)* 128A(2): 245-253; 1977.

Serological markers of hepatitis B virus (HBV) were detected and analyzed in 291 patients with primary liver carcinoma (PLC), 39 with hepatitis B, and 35 with cirrhosis; 143 individuals (100 normal blood donors and 43 patients with non-hepatic cancers) served as controls. Antigens for the viral coat (HBsAg), "e"/anti-"e" system (HBeAg), and core antibody (HBc) served as the serological markers. AntiHBc and  $\alpha$ -fetoprotein were detected by counter-electrophoresis, HBsAg and anti-HBs by solid-phase radioimmunoassay, and HBeAg and anti-HBe by radial immunodiffusion. HBsAg was found in 12%, anti-HBs in 38%, and anti-HBc in 26% of the controls. Anti-HBs was high in patients suffering from hepatitis B (84.6%), cirrhosis (71.4%), and PLC (51.9%). Anti-HBc was present at high levels also (100%, 88.6%, 87.3%, respectively), but only during the replication phase of HBV infection. On the contrary, anti-HBs was higher in the control groups (38% blood donors, and 32.5%, cancer patients) than in the HBV-infected groups (2.5%, 17.1%, and 16.1%). It is concluded that this evidence implicates HBV in the etiology of PLC. (15 refs.)

**77-2858 Oral Contraceptives and Benign Liver Tumours.** (Eng.) Vessey, M. P. (Dept. Social and Community Medicine, Oxford, England) Kay, C. R.; Baldwin, J.



A.; Clarke, J. A.; Macleod, I. B. *Br Med J* 1(6068): 1064-1065; 1977.

Several data bases were used to search for cases of benign liver tumor in women of childbearing age in order to determine the effects of oral contraceptives on the incidence of these tumors. No cases of benign liver tumor were identified in either the Royal College of General Practitioners oral contraceptive study, the Oxford Family Planning Association follow-up study of women using different methods of contraception, or the Oxford record linkage study. The Scottish national diagnostic index, which is concerned with hospital admissions and deaths occurring in a population of approx 5 million, showed one case of benign liver tumor in a woman of childbearing age in the years of 1968-1974. The patient was a 23-yr-old woman who had been taking oral contraceptives for about 2 yr. The pathological diagnosis was hamartoma. Benign tumors of the liver are extremely rare in both users and nonusers of oral contraceptives. (5 refs.)

77-2859 **Hepatoblastoma in Infant Sister and Brother.** (Eng.) Napoli, V. M. (Clinical Lab., Grady Memorial Hosp., 80 Butler St., S. E., Atlanta, GA 30303) Campbell, W. G. *Cancer* 39(6): 2647-2650; 1977.

The case histories of a 15-mo-old girl with motor retardation and hepatoblastoma and her 15-mo-old brother with cryptorchidism and hepatoblastoma are presented. A family study failed to provide any conclusive data on the etiology in these siblings. (15 refs.)

77-2860 **Hyaline Cytoplasmic Inclusions in Human Hepatoma: A Case Report.** (Eng.) Grimelius, L. (Dept. Clinical Cytology, Univ. Hosp., S-750 14 Uppsala, Sweden) Stenram, U.; Westman, J.; Westman-Naeser, S. *Acta Cytol (Baltimore)* 21(3): 469-476; 1977.

The case of a 79-yr-old woman with hepatoma is reported. The patient had an enlarged liver and a pathological liver scintigram. Percutaneous liver biopsies were performed both with Menghini- and fine needles. The most prominent feature was the presence of hyaline cytoplasmic PAS-negative inclusions in the liver parenchymal cells. There was no nuclear atypia. Electron microscopy disclosed two types of cytoplasmic changes. One consisted of a lamellar ultrastructure and was interpreted as a hyperplasia of smooth-surface endoplasmic reticulum. The other change consisted of smooth, globular, nonmembrane-limited regions containing amorphous or finely fibrillar material. This was interpreted as corresponding to the hyaline inclusions visible in the light microscope. The presence of PAS-negative hyaline cytoplasmic inclusions may thus be a sign of hepatoma. This may be of relevance for the diagnostic considerations of material obtained by fine-needle biopsy. (25 refs.)

77-2861 **Liver Metastasis of Gastric Carcinoma after Radical Operation.** (Jpn.) Furukawa, H. (Dept. Surgery, Center Adult Diseases, Osaka, Japan) Iwanaga, T.; Tanaka, H.; Koyama, H. *Jpn J Cancer Clin* 23(3): 198-202; 1977.

Following radical surgery for the treatment of gastric cancer, liver metastasis developed in 75/1178 patients. The primary lesions had the gross appearance of Borrmann's type 2, and they occurred predominantly in the antrum. Histologic studies showed well-differentiated adenocarcinoma with lymphatic metastasis and vascular invasion invading beyond the submucosal layer. SGOT, SGPT, alkaline phosphatase, and lactic dehydrogenase were often elevated before the recurrence was manifested clinically. (20 refs.)

77-2862 **Primary Carcinomas of the Stomach with Association of Carcinoma of Other Organs.** (Jpn.) Kishimoto, H. (First Dept. Surgery, Sch. Medicine, Tottori Univ., Japan) Oku, H.; Sugihara, T.; Fujii, T.; Yoshida, Y.; Yoshioka, T.; Tanaka, K.; Kawaguchi, H.; Miyano, Y.; Maeta, M.; Andachi, H.; Koga, S. *Jpn J Cancer Clin* 23(6): 550-556; 1977.

Between 1948 and 1975, 18/1,447 cases of primary gastric cancer were associated with primary cancer of another organ. Five of these were synchronous double primary cancers, and eight developed after gastrectomy. Five patients underwent gastrectomy several yr after treatment of the first cancer. One of the 18 cases was a triple cancer. (20 refs.)

77-2863 **Factors Influencing the Postoperative Free Interval of Gastric Cancer.** (Jpn.) Kusama, S. (First Dept. Surgery, Faculty Medicine, Univ. Tokyo, Tokyo, Japan) *Stomach Intest* 12(1): 61-72; 1977.

The time of recurrence was analyzed statistically in 3,419 patients with recurrent gastric cancer. The postoperative free interval showed a log-normal distribution. The time of recurrence had a statistically significant relationship to the site of the recurrence, duration of symptoms, survival, patient age, site and macroscopic size of the gastric cancer, whether it was an early or late gastric cancer, Borrmann's classification, histological classification, amount of infiltrative growth, degree of extension and penetration, degree of lymph node metastasis at the time of gastrectomy, classification of gastric resection based on the extent of lymph node removal, adjuvant chemotherapy, and the existence of simultaneous multiple gastric cancers at the time of gastrectomy. It is suggested that factors related to the time of recurrence of gastric cancer have an close relationship to the doubling time of the gastric cancer. (1 ref.)

- 77-2864 **Cellular Leiomyomas of the Stomach in 49 Patients.** (Eng.) Appelman, H. (Dept. Skin and Gastrointestinal Pathology, Armed Forces Inst. Pathology, Washington, DC 20306) Helwig, E. B. *Arch Pathol Lab Med* 101(7): 373-377; 1977.

Gastric cellular leiomyomas that occurred in 49 patients (37 men and 12 women, mean age 57.2 yr) over a 21-yr period were examined histologically. The results show that the tumors are composed of tightly packed, generally uniform spindle cells arranged in palisades, whorls, or interdigitations. Although these neoplasms have been variably considered to arise from gastric smooth muscle or Schwann cells, their morphogenesis has not been resolved completely. They may derive from a multipotential gastric stromal stem cell that is capable of differentiating toward smooth muscle. A cellular gastric tumor composed of fairly uniform, elongated spindle cells is unlikely to be malignant. Of the 49 tumors, only one metastasized, a 17-cm neoplasm with a mitotic count of 5/50 high-power fields. The combination of an increased mitotic rate and large tumor size is possibly indicative of malignant potential. (18 refs.)

- 77-2865 **Prospective and Retrospective Studies for Long Years on the Cancerous Change from the Gastric Ulcer with Fiberscopic Biopsy and Gastrocamera Pictures.** (Eng.) Oguro, Y. (Dept. Internal Medicine, Natl. Cancer Center Hosp., Tsukiji 5-chome, Chuo-ku, Tokyo, 104 Japan) Yoshida, S. *Gastroenterol Jpn* 12(1): 44-51; 1977.

Prospective and retrospective studies were conducted to determine if there is an association between benign gastric ulcer and gastric carcinoma. In the prospective study, 239 cases of benign gastric ulcer were followed by repeated fiberscopic biopsy for > 2 yr. Five of the 239 cases (2.1%) developed gastric carcinoma. The av period from the initial negative biopsy to the positive one was 3 yr 5 mo, the longest period being 4 1/2 yr and the shortest 2 yr. There were 3 differentiated adenocarcinomas, 1 poorly differentiated adenocarcinoma, and 1 signet ring cell carcinoma. In another study, gastrocamera films going back > 2 yr were available for 43 patients having confirmed gastric malignancies after surgery. There were five cases of gastric cancer arising at ulceration sites that could be traced back for > 10 yr. These cases are considered premalignant lesions. (10 refs.)

- 77-2866 **Neuroendocrine Carcinomas of the Gastrointestinal Tract (Meeting Abstract).** (Eng.) Chejfec, G. (Hines VA Hosp., Hines, IL 60612) Gould, V. E. *Lab Invest* 36(3): 333; 1977. (no refs.)

- 77-2867 **Carcinogenesis (Letter to Editor).** (Eng.) Dunning, R. E. (11 Hatfield St., Green Point, Cape Town, South Africa) *S Afr Med J* 52(2): 51; 1977.

A case is reported in which habitual rapid ingestion of extremely hot beverages may have been a causal factor in gastric cancer. A contributory factor was the wearing of dentures preventing the sensory nerves of the palate from rejecting the hot liquid. (1 ref.)

- 77-2868 **Realization of Genetic Information Programming Synthesis of Pepsinogen-Pepsin in the Gastric Mucosa and Tumors of the Human Stomach.** (Rus.) Seits, I. F. (N. N. Petrov Inst. Oncology, Ministry Public Health USSR, Leningrad, USSR) Kalinovskii, V. P. *Vestn Akad Med Nauk SSSR* (3): 61-64; 1977.

Analysis of the gastric mucosa of patients with carcinoma, ulcers or polyposis of the stomach indicated 19 to 20 individual forms of RNA. Pepsinogen-pepsin was synthesized by the bound polysomes of the gastric mucosa of the stomach from ulcer and polyposis patients, but not by those of cancerous stomach. (13 refs.)

- 77-2869 **Extragenital Mixed Heterologous Tumor of Mullerian Type Arising in the Cecal Peritoneum: Report of a Case.** (Eng.) Weisz-Carrington, P. (New York Univ. Medical Center, Sch. Medicine, 550 First Ave., New York, NY 10016) Bigelow, B.; Schinella, R. A. *Dis Colon Rectum* 20(4): 329-333; 1977.

A case report is presented of a 77-yr-old woman with a heterologous mixed Mullerian tumor that arose in the cecal serosa. There was no history of endometriosis, nor was the condition demonstrated histologically. Evidence of a mesothelial origin was found microscopically in the adjacent cecal peritoneum. All heterologous mixed Mullerian tumors previously reported in the medical literature have originated only in the genital system. (5 refs.)

- 77-2870 **A Case of Cancer of the Ileum Complicating Tuberculosis in the Same Lesion.** (Jpn.) Noda, S. (Dept. Surgery, Natl. Fukuoka Central Hosp., Fukuoka, Japan) Lui, K.; Ushijima, K.; Matuura, R.; Ikejiri, T.; Nakamura, Y.; Watanabe, H. *Jpn J Cancer Clin* 23(3): 240-244; 1977.

The history of a 58-yr-old woman with two tuberculous lesions of the ileum, a lesion with coexisting tuberculosis and cancer of the ileum, and an ileocecal cancer is presented. In the lesion with tuberculosis and cancer of the ileum, a well-differentiated adenocarcinoma and a tubercle with caseous and necrotic degeneration were present. Immunological reactions such as lymphocyte infiltration surrounding the cancer were not observed. (22 refs.)



- 77-2871 **Malignant Neoplasms of the Hematopoietic Systems in Three Mutants of *Drosophila melanogaster*.** (Eng.) Gateff, E. (Biologisches Institut I, Zoologie, Albert-Ludwigs-Universität, 78 Freiburg i. Br., Schanzlestr. 9, W. Germany) *Ann Parasitol Hum Comp* 52(1): 81-83; 1977.

Three ethyl methanesulfonate-induced larval, recessive-lethal mutations of *Drosophila melanogaster* caused malignant neoplasms of the phagocytic precursor cells. One mutation affected the control mechanism of blood cell production, while the other two prevented the differentiation of phagocytic blood cells at an early developmental stage, causing an accumulation of immature blood cells in the hemolymph. *Drosophila* hematopoietic mutants may shed light upon the genetics of the neoplastic transformation of blood cells. (3 refs.)

- 77-2872 **Properties of Teratocarcinoma-Thymus Somatic Cell Hybrids.** (Eng.) Miller, R. A. (Dept. Human Genetics, Yale Univ., New Haven, CT 06520) Ruddle, F. H. *Somatic Cell Genet* 3(3): 247-261; 1977.

The resemblance of teratocarcinoma-thymus hybrid cells (PCT hybrids) to the embryonal carcinoma parent was examined by phase contrast and electron microscopy. Both embryonal carcinoma and PCT hybrid lines, shown previously to give rise to multidifferentiated tumors and hence to be pluripotent, were morphologically similar. Both had high levels of alkaline phosphatase. Like embryonal carcinoma cells, PCT hybrids did not express Thy 1 alloantigen (which is present on thymocyte parental cells). However, PCT hybrids did exhibit H2 antigens, which were present only at very low levels, if at all, on embryonal carcinoma cells. (39 refs.)

- 77-2873 **Karyotype Analysis of Teratocarcinomas and Embryoid Bodies of C3H Mice.** (Eng.) Iles, S. A. (Dept. Zoology, South Parks Road, Oxford, OX1 3 PS, England) Evans, E. P. *J Embryol Exp Morphol* 38: 77-92; 1977.

Four transplantable teratocarcinomas (Nos. 17, 86, 106, and 145) and embryoid bodies derived from C3H mice were studied over several transplant generations using G-banding techniques to identify the karyotypes. Ip transfer of all four teratocarcinomas resulted in the formation of ascitic fluid containing embryoid bodies. Chromosome studies were carried out on various transplant generations of both the solid tumors and their embryoid bodies. Chromosome counts for the early transplant generations showed movement from a diploid to a hyperdiploid number of chromosomes in tumor 86 and bifurcation into two sublines in tumor 145; tumor 106 had abnormal chromosome counts as early as the third generation. Tumor 17 and subline a of tumor 145 had normal karyotypes, and they were able to differentiate into a wide

range of tissues. Tumors 86 and 106 and subline b of tumor 145, which had karyotypic abnormalities, showed almost identical restrictions in their capacity for differentiation. Banding techniques showed that trisomies were largely responsible for the abnormal chromosome counts. In all cases, changes in karyotype were associated with restriction of differentiation. (21 refs.)

- 77-2874 **Increased Expression of Actin-like Contractile Protein in Preneoplastic and Neoplastic Lesions in Rat Liver.** (Eng.) Toh, B. H. (Dept. Pathology and Immunology, Monash Univ. Medical Sch., Melbourne, Victoria, 3181, Australia) Cauchi, M. N.; Cook, P. C.; Muller, H. K. *Br J Cancer* 35(6): 761-767; 1977.

Cryostat sections of 16 preneoplastic and 14 neoplastic hepatic lesions induced in Wistar rats fed 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB, 0.06%) were examined by indirect immunofluorescence with human serum containing smooth-muscle antibody (SMA). In areas of nodular hyperplasia, preneoplastic lesions showed strong cytoplasmic staining of proliferating oval cells and of the cell outlines of hepatocytes. In carcinomas, poorly differentiated hepatocytes showed staining of cell outlines, but well-differentiated tumor cells forming glandular structures showed only staining of the luminal surfaces. The stromal cells also showed cytoplasmic staining. Morphologically normal areas of 3'-Me-DAB-treated livers showed weak staining of cell outlines, similar to normal liver. The specificity of the staining reactions was established by failure of the staining in parallel control sections treated with (1) normal human serum, (2) SMA serum neutralized by absorptions with homogenates of smooth muscle, or (3) extracts of actin. The results suggest that there is an increased expression of actinlike contractile protein in preneoplastic and poorly differentiated neoplastic liver cells. (23 refs.)

- 77-2875 **Electron Microscopic Studies of Mammalian Cells Stained with Platinum-Pyrimidine Blues.** (Eng.) McAllister, P. K. (Dept. Biochemistry, Michigan State Univ., East Lansing, MI 48824) Rosenberg, B.; Aggarwal, S. K.; Wagner, R. W. *J Clin Hematol Oncol* 7(2): 717-725; 1977.

A variety of mammalian cell types were stained with platinum-pyrimidine blues and examined electron microscopically to clarify the nature of the materials stained. Cells were obtained from continual cell lines, primary cultures, or directly from animals. The cells were fixed in 2% glutaraldehyde in 50 mM cacodylate buffer at pH 7.4 for 2 hr. After fixation, the cells were resuspended in 1% platinum-thymine blue or platinum-uracil blue for 1 hr. In some cases cells were postfixed in 1% osmium tetroxide prior to staining. Examination showed staining of the ribosomes, cell surfaces, chromatin, and nucleolus. There was no difference in staining between tumorigenic and nontumorigenic cells. Glutaraldehyde

concentration and type of buffer had no effect on the stains. Cells postfixed with osmium tetroxide and stained with platinum-pyrimidine blues displayed increased contrast compared with cells fixed with either heavy metal alone. S-180 ascites cells were treated with enzymes following fixation in an attempt to characterize the surface staining material. Pronase and neuraminidase greatly reduced the amount of surface stain, but deoxyribonuclease, ribonuclease, and hyaluronidase had no effect. (2 refs.)

**77-2876 Ultrastructural Comparison of Mast Cells and Mastocytoma Cells in the Mouse (Meeting Abstract).** (Eng.) Murata, F. (Dept. Anatomy, Shinshu Univ. Sch. Medicine, Matsumoto, Japan) Momose, Y.; Nagata, T. *J Electron Microsc (Tokyo)* 25(3): 197; 1976. (no refs.)

**77-2877 The Cytoskeleton and Cell Transformation to Malignancy. Microtubules, Microfilaments and Growth Properties In Vitro.** (Eng.) Brinkley, B. R. (Div. Cell Biology, Dept. Human Biological Chemistry and Genetics, Univ. Texas Medical Branch, Galveston, TX) Miller, C. L.; Fuseler, J. W.; Pepper, D. A.; Wible, L. J. *Cancer Bull* 29(1): 13-15; 1977.

The extensive cytoplasmic microtubule complex identified in normal cells in vitro was consistently undetectable or greatly diminished in transformed cells. Since one of the functions of the cytoskeleton appears to be the regulation of surface membrane organization and distribution of surface receptors, the alteration in the cellular microtubules and microfilaments could be fundamental to malignancy, in particular to the increased growth capacity of transformed cells. (1 ref.)

**77-2878 Comparative Study of a Low-cancer Mouse Cell Line and its High-cancer Derivative.** (Eng.) Le-Francois, D. (Tissue Culture Virology Lab., E.R. C.N.R.S. No. 38, Institut Gustave-Roussy, 94800 Villejuif, France) Leon, B.; Barski, G. *J Microsc Biol Cell* 27(1): 25-32; 1976.

A comparative study was performed on a low-malignancy C57B46 mouse cell line (P4bis) and its highly malignant derivative (P4bisT). P4bisT was derived from an in vitro culture of an in vivo induced tumor of P4bis. Both lines were tested for differences in tumor-producing capacity in the cheek pouch of cortisone-treated Syrian hamsters, the chorioallantoic membrane, and the brain of chick embryos. In the cheek pouch, P4bis produced no growth with inocula of  $4$  to  $5 \times 10^6$  cells; 100% growth was observed with inoculation of  $2.5 \times 10^6$  P4bisT cells. Similar results were observed in the chorioallantoic membrane of embryonated chick eggs and the brain of 11-day chick embryos. The av number of chromosomes in P4bisT was 60 to 70 while that in P4bis was 80 to 90. The differences in tumorigenicity were probably due to intrinsic differences in the cell lines. In vitro, the P4bis cells

displayed a fairly regular growth pattern with few cell overlappings. The P4bisT cells had an irregular pattern of growth with frequent overlappings. The saturation density for the P4bis line was  $1.8 \times 10^5$  cells/cm<sup>2</sup>; that for P4bisT was  $5.5 \times 10^5$ ; the av doubling times were 26 and 12 hr, respectively. Electron microscopy of the cell lines demonstrated no C-type virus particles in the P4bis line but particles in the P4bisT line. Although no evidence of infectious viruses was obtained with XC cells in the P4bis line, the tests were positive in the P4bisT line between passages 85 and 116. (35 refs.)

**77-2879 Experimental Studies on the Hematogenous Metastasis of Tumors in Mice Under Hyperbaric Oxygenation.** (Jpn.) Umemura, H. (2nd Dept. Surgery, Kinki Univ., Sch. Medicine, Sayama-cho, Minamikawachigun, Osaka, 589, Japan) *Arch Jpn Chir* 46(2): 121-134; 1977.

The effects of hyperbaric oxygenation (HO) on tumor metastasis and host survival were studied in DDD mice bearing Ehrlich ascites carcinoma and C3H mice bearing MH 134 ascites hepatoma. Mice inoculated with tumor cells ( $3 \times 10^5$  /0.05 ml, iv) just before exposure to HO had a high frequency of pulmonary metastases and a shortened survival compared with animals inoculated 24 to 48 hr before exposure. Subsequent in vivo and in vitro experiments showed that 100% oxygen at 4 or 2 atmospheres absolute (ATA) for 2 hr had no significant effect on the number of dead or viable cells in tumors transplanted into the peritoneal cavity. However, tumor cell emboli were observed more often in the small arterioles or capillaries of rats exposed to 100% oxygen at 2 ATA than in those of animals exposed to air at atmospheric pressure. The tumor emboli consisted of clumps or fragments of solitary cells. These results show that the enhancement of hematogenous metastasis to the lung in rats inoculated before HO was due to microcirculatory changes or alveolar damage caused by the oxygenation. (53 refs.)

\* (Rev): 77-2431, 77-2435, 77-2438, 77-2439, 77-2443, 77-2444.

\* (Chem): 77-2446, 77-2477, 77-2483, 77-2500, 77-2502, 77-2521, 77-2544, 77-2548, 77-2549, 77-2550, 77-2551, 77-2553, 77-2561, 77-2563, 77-2568, 77-2574, 77-2579, 77-2580, 77-2581, 77-2588, 77-2589, 77-2590, 77-2591, 77-2598.

\* (Phys): 77-2631.

\* (Viral): 77-2655, 77-2660, 77-2689, 77-2690.

\* (Immun): 77-2714, 77-2766, 77-2770, 77-2771, 77-2800, 77-2812.

\* (Epid): 77-2892, 77-2893, 77-2897.



## EPIDEMIOLOGY AND BIOMETRY

- 77-2880 **Some Aspects of Geocancerology.** (Eng.) Verhasselt, Y. (Geografisch Instituut Vrije, Universiteit Brussel, Brussels, Belgium.) *Med Biol Environ* 4(1): 75-77; 1976.

Two geographical approaches to the study of environmental carcinogenesis are discussed. The first is the use of distribution maps to identify spatial correlations and associations between cancer and causal factors; the second is the application of multivariate techniques to hypotheses correlating cancer with environmental factors. (9 refs.)

- 77-2881 **Geographic Distribution of Cancer in Middle Eastern Countries.** (Eng.) Racoveanu, N. T. (WHO Eastern Mediterranean Region Office, Alexandria, Egypt) *Med Biol Environ* 4(1): 36-50; 1976.

The relative incidence of tumors by histological type was analyzed for: Afghanistan, Egypt, Iran, Iraq, Israel, Lebanon, Pakistan, Sudan, and Tunisia. Particular discussion is made of the occurrence of lymphomas, oral cancer, nasopharyngeal carcinoma, and cancers of the bladder, breast, lung, uterine cervix, larynx, stomach, and colon. A very high frequency of bone tumors, mainly osteosarcomas, is found in Pakistan and Afghanistan: in Peshawar province, Pakistan, the incidence of this tumor is 8.9% of all tumors in men. There is a high incidence of eye tumors in Afghanistan and Sudan; this has been attributed to high solar radiation, dust, and other irritative factors in a dry and hot climate. (28 refs.)

- 77-2882 **Environmental Influence on Cancer Incidence.** (Eng.) Rose, E. (Bantu Cancer Registry, East London, S. Africa) *Med Biol Environ* 4(1): 51-59; 1976.

The incidence of esophageal cancer was estimated on a district-to-district basis in the Transkei, South Africa, in an attempt to determine possible etiological factors. The standardized incidence rates for some districts are the highest in the world, but in others they are low: the variation between districts is as much as 116 and 3 in men and 20 and 2 in women (1955-1959). The pattern of variation among districts did not vary significantly from 1955-1969. Variations in incidence have been considered with regard to population density, topography, geology, veld type, soil type, and rainfall. Most of the people in the high incidence areas exist by subsistence farming. An inadequate diet, with lack of essential elements or other protective factors, may be involved in the development of the disease. (no refs.)

- 77-2883 **Human Monitoring Program in Occupational Contamination by Chemicals in Rural Medicine.** (Eng.) Fournier, P. E. (Hopital Fernand WIDAL, 200 rue de Faubourg Saint Denis, 75475 Paris Cedex 10, France) Efthymiou, M. L.; Dally, S. *J Occup Med* 19(7): 464-468; 1977.

Based on a toxicity study of organochloride and organophosphate pesticides, a three-step study is recommended for evaluation of the hazard of chemicals to man. These steps include clinical observation, occupation data collection, and a systematic study of av human groups. (19 refs.)

- 77-2884 **Nitrate Fertilizers as Environmental Pollutants: Positive Correlation Between Nitrates ( $\text{NaNO}_3$  and  $\text{KNO}_3$ ) Used per Unit Area and Stomach Cancer Mortality Rates.** (Eng.) Zaldivar, R. (2170 N.W. 11th St., Suite 53, Miami, FL 33125) *Experientia* 33(2): 264-265; 1977.

Studies on the use of nitrate fertilizers in Chile indicated a significant correlation between the use of nitrates and stomach cancer mortality rates. It is suggested that nitrogen fertilizers not be used for 10 yr in order to restore a balance to nature. (11 refs.)

- 77-2885 **Some Problems of Primary (Hygienic) Prophylaxis of Oncological Diseases.** (Rus.) Beliaev, I. I. (Gorki Medical Inst., Gorki, USSR) Shkodich, P. E.; Gracheva, M. P. *Vestn Akad Med Nauk SSSR* (2): 16-22; 1977.

The organic synthesis of polycyclic aromatic hydrocarbons and the resulting contamination of the air, water, soil and vegetation are discussed. Recommendations for the control of these hazardous conditions are presented. (32 refs.)

- 77-2886 **Environmental Asbestos Pollution Related to Use of Quarried Serpentine Rock.** (Eng.) Rohl, A. N. (Environmental Sciences Lab., Mount Sinai Sch. Medicine, City Univ. New York, New York, NY 10029) Langer, A. M.; Selikoff, I. J. *Science* 196(4296): 1319-1322; 1977.

Data are presented on the concentrations of airborne chrysotile asbestos in the vicinity of roads surfaced by quarried serpentine crushed rock near Rockville, Maryland. The concentrations were approx 1,000 times greater than the av values measured in 49 U.S. cities. (19 refs.)

77-2887 **Styrene Butadiene Rubber Synthetic Plants and Leukemia (Letter to Editor).** (Eng.) Smith, A. H. (Occupational Health Studies Group, Sch. Public Health, Univ. North Carolina, Chapel Hill, NC) Ellis, L. *J Occup Med* 19(7): 441; 1977.

Neither styrene nor butadiene, two monomers used in the synthesis of rubber, have been found to be carcinogenic, although styrene oxide has caused malignant lymphoma in mice. The five deaths reported in one synthetic plant may have been the result of occupational exposure to other chemicals as well, since each death resulted from a different neoplasm of the lymphatic and hematopoietic tissue. (3 refs.)

77-2888 **False-Positive and False-Negative Rates for Carcinogenicity Screens.** (Eng.) Fears, T. R. (Office Field Studies and Statistics, NCI, Bethesda, MD 20014) Tarone, R. E.; Chu, K. C. *Cancer Res* 37(7): 1941-1945; 1977.

Some chemical carcinogen screening programs might have high false-positive error rates. With designs presently used at NCI and historical spontaneous tumor rates based on control animals in previous experiments, the writer computed upper bounds on the false-positive error rates for several screening strategies. False-positive results are much less likely to occur at tissue sites with low spontaneous tumor rates; hence, the site at which a significant tumor increase occurs is important. There is danger in relying solely on the finding of statistical significance without incorporating biological knowledge and corroborative evidence such as the presence of a dose-response relationship or experimentally consistent results in different species or sexes. A report by the NCI Carcinogenesis Program illustrates these concepts. (3 refs.)

77-2889 **Hazards from Plutonium Toxicity.** (Eng.) Cohen, B. L. (Univ. Pittsburgh, Pittsburgh, PA 15260) *Health Phys* 32(5): 359-379; 1977.

Using standard models and theories, the number of cancer fatalities caused by the dispersal of plutonium can be estimated. This process includes development of estimates of Pu toxicity by inhalation and ingestion, calculation of meteorological dispersal by the Gaussian plume model, estimates of resuspension effects from empirical models, and estimation of long-term effects from a comparison with natural uranium in soil. One cancer can be expected for each 200  $\mu$ g of reactor Pu (2.5 g/Ci) inhaled or for each 1.0 g ingested. For dispersal in a city, there will be one eventual fatality per 18 g of reactor Pu dispersed; this effect is dominated by inhalation from the initial cloud, with early resuspension effects somewhat less important and long-term effects essentially negligible. (74 refs.)

77-2890 **Environmental Selenium and Human Cancer Mortality (Meeting Abstract).** (Eng.) Shamberger, R. J. (Cleveland Clinic, Cleveland, OH 44106) *Proc Am Assoc Cancer Res* 18: 167; 1977. (no refs.)

77-2891 **A Multidisciplinary Approach to Thyroid Carcinoma Developing in Irradiated Patients (Meeting Abstract).** (Eng.) Khandekar, J. D. (Evanston Hosp. (EH) Ev. 60201 and Northwestern Univ. Medical Sch., Chicago, IL) Scanlon, E. F.; Murphy, E. D.; Garces, R. M.; Swelsted, J. *Proc Am Assoc Cancer Res* 18: 59; 1977. (no refs.)

77-2892 **Clinico-Epidemiological Characteristics of Chronic Lymphatic Leukemia (Meeting Abstract).** (Rus.) Plotnikov, Iu. K. (1st Dept. Therapy, D. I. Ul'ianov Kuibyshev Medical Inst., Kuibyshev, USSR) *Vopr Onkol* 22(12): 69; 1976. (no refs.)

77-2893 **Mumps Orchitis and Testicular Tumours (Letter to Editor).** (Eng.) Ehrengut, W. (Hamburg, W. Germany) Schwartau, M. *Br Med J* 2(6080): 191; 1977.

The review of a series of 294 patients with testicular tumors indicated that mumps orchitis is seldom found in the past history of such patients. However, previous epididymitis was found in the affected testicle in 4.9% of the patients. Out of 87 patients who had mumps orchitis between 1960 and 1970, none developed a testicular tumor. (2 refs.)

77-2894 **Epidemiologic Study of Prostatic Cancer: Preliminary Report.** (Eng.) Schuman, L. M. (Program in Epidemiology, Univ. Minnesota, Sch. Public Health, Minneapolis, MI 55455) Mandel, J.; Blackard, C.; Bauer, H.; Scarlett, J.; McHugh, R. *Cancer Treat Rep* 61(2): 181-186; 1977.

Preliminary data on 53 patients in a projected study population of 200 white patients with prostatic cancer provide some evidence to support both the sexual activity and venereal transmission hypotheses. In contrast to hospitalized and/or neighborhood controls, the cancer patients show greater proportions of selected sexual activities compatible with venereal transmission of an infectious agent, such as number of sexual partners, use of prostitutes, prior venereal disease, and genital infections in the spouse. The patients also appear to have had higher fertility and more prostatic cancer in blood relatives than controls. Age at first intercourse and at first marriage are lower among the cancer patients than among the controls. Antibody titrations for herpesvirus and cytomegalovirus, although they do not reveal striking disparities in positivity, tend to show higher titers among the cancer cases. (30 refs.)



- 77-2895 **Studies in the Epidemiology of Prostatic Cancer: Expanded Sampling.** (Eng.) Rotkin, I. D. (Univ. Illinois Coll. Medicine, Chicago, IL 60612) *Cancer Treat Rep* 61(2): 173-180; 1977.

As a result of comparison of 111 prostatic cancer patients with matched control patients for selected risk variables, the patients are characterized by three main trends: delayed sexual drive and development, early repression of sexuality, and premature cessation of sexuality. Excessive numbers of patients reported occupational exposure to fertilizers and auto exhaust fumes. Diets of the patients were higher in animal fats. There were no differences between both groups in frequencies of multiple marriages or sex partners or stressful effects from selected events early or late in life. Trends for circumcision and other variables are presented. The data suggest that early differences are hormonally conditioned, they support a provisional endogenous rationale for initiation of prostatic cancer, and they oppose a hypothesis favoring transmissible oncogenic agents. If the results continue to hold up with increased sampling, limitation of sexual activity at any time of life may increase risk. (3 refs.)

- 77-2896 **Race, Socioeconomic Status, and Prostatic Cancer.** (Eng.) Ernster, V. L. (Sch. Public Health, Univ. California, Berkeley, CA 94720) Winkelstein, W.; Selvin, S.; Brown, S. M.; Sacks, S. T.; Austin, D. F.; Mandel, S. A.; Bertolli, T. A. *Cancer Treat Rep* 61(2): 187-191; 1977.

Mortality and incidence data from Alameda County, California, were used in an attempt to determine whether the higher occurrence of prostatic cancer among black men compared with whites in the US might be explained by racial differences in factors associated with socioeconomic status. Each death or case of prostatic cancer was assigned to a social class based on the census tract of residence, and rates by race and socioeconomic status were computed. Comparison of age-specific mortality and incidence rates by socioeconomic status reveals no gradient in either whites or blacks. The higher risk for blacks hold up at almost every age and socioeconomic level. However, the racial differences are less pronounced for incidence than for mortality. Racial differences in cases in which prostatic cancer was listed as related to, but not the primary cause of, death are also examined. (12 refs.)

- 77-2897 **Blood Group Distribution in Prostatic Cancer Patients.** (Eng.) Wajsman, Z. (Roswell Park Memorial Inst., Buffalo, NY) Saroff, J.; Murphy, G. P. *J Surg Oncol* 9(3): 289-291; 1977.

Blood group distribution was analyzed in 264 Stage D relapsing patients from the National Prostatic Cancer Project. No significant differences were found in the distribution of blood groups in the prostatic cancer patients compared with the control population: 35.98% of the patients were Group A,

47.35% Group O, 12.5% Group B, and 4.17% Group AB. The corresponding percentages for the controls were 40.77%, 45.55%, 9.9%, and 3.72%. This study does not support the suggested relationship between blood Group A and susceptibility to carcinoma of the prostate. Other patient groups may however, be of value in investigations for this relationship (9 refs.)

- 77-2898 **Age Distribution of Condyloma Acuminatum and of Carcinoma of the Vulva and Penis in Uganda (Meeting Abstract).** (Ger.) Schmauz, R. (Lubeck, W. Germany) Owor, R. *Zentralbl Allg Pathol* 121(3): 299; 1977. (no refs.)

- 77-2899 **Age-specific Incidence Rates of Endometrial Cancer (Letter to Editor).** (Eng.) Casey, M. J. (Univ. Connecticut Sch. Medicine, Farmington, CT) *JAMA* 238(3): 213; 1977.

A review of cases of endometrial cancer reported between 1953 and 1973 indicated a significant increase in the diagnosis of the neoplasm in the age groups at risk and an increasing trend toward diagnosis in the past 20 yr. Future studies should report the age-specific incidence. (3 refs.)

- 77-2900 **Endometrial Cancer and Extraglandular Oestrogen Biosynthesis.** (Eng.) Schindler, A. E. (Universitäts-Frauenklinik, D-7400 Tübingen, W. Germany) *Geburtshilfe Frauenheilkd* 37(3): 242-251; 1977.

Persons at risk for endometrial cancer include older women and women with menstrual disorders, reduced fertility, obesity, diabetes, hypertension, hirsutism, and hyperplasia of the ovarian stroma or hilus cells. Many of these characteristics can be explained by the extraglandular, peripheral aromatization of androgens to estrogens, particularly the conversion of androstenedione to estrone. This is supported by an increased plasma estrone/estradiol ratio and increased conversion with age and overweight. In vivo and in vitro investigations have demonstrated the participation of adipose tissue in peripheral estrogen production. The compiled data indicate that extraglandular estrogen production, by affecting the endometrium over long periods of time, is an important etiologic factor in endometrial cancer. In the absence of normal cyclic changes of estradiol and progesterone, estrogen can subject the endometrium to significant hormonal stress. Pre- and postmenopausal estrogen therapy should, therefore, be critically reevaluated. (162 refs.)

- 77-2901 **Marital Status and Incidence of Ovarian Cancer: The U.S. Third National Cancer Survey, 1969-71.** (Eng.) Weiss, N. S. (Fred Hutchinson Cancer Res.

Center, 1124 Columbia St., Seattle, WA 98104) Young, J. L.; Roth, G. J. *J Natl Cancer Inst* 58(4): 913-915; 1977.

Between 1968-1971 the incidence of ovarian cancer was 60%-70% higher in American women who were never married than in those who were married or had married at one time. This relationship was present in both whites and blacks and in all age groups over 25 yr. Among ovarian epithelial tumors, those for which the incidence rates differed the most were endometrioid and clear cell tumors. The incidence of tumors of germ cell and sex cord-mesenchymal origin showed no relationship to marital status. (17 refs.)

**77-2902 Incidence of the Histologic Types of Ovarian Cancer: The U.S. Third National Cancer Survey, 1969-1971.** (Eng.) Weiss, N. S. (Dept. Epidemiology, Univ. Washington, Seattle, WA 98195) Homonchuk, T.; Young, J. L. *Gynecol Oncol* 5(2): 161-167; 1977.

Using data gathered in the US Third National Cancer Survey, 1969-1971, the incidence of the major histologic types of primary ovarian cancer, as well as the variation in that incidence according to several demographic characteristics, was examined. All types of ovarian cancer except germ cell tumors increased in incidence until the seventh and eighth decades of life, after which the rates plateaued. Epithelial tumors were equally common in young whites and blacks, but they were substantially more common in whites among middle-aged and older women. The incidence of nonepithelial ovarian tumors was similar in the two races. Rates of epithelial tumors were greatest in single women, but nonepithelial tumor occurrence showed no correlation with marital status. There was little variation among the ovarian epithelial tumors in their relationship to age, race, and marital status, although true associations may have been obscured because of classification inconsistencies in the data. (8 refs.)

**77-2903 Some Epidemiologic Observations on the Magnitude and Nature of the Breast Cancer Problem.** (Eng.) Cutler, S. J. (Wayne State Univ. Sch. Medicine, Michigan Cancer Foundation, Detroit, MI 48201) *Cancer Detect Prevent* 1(2): 245-254; 1976.

Trends in the incidence, mortality, and patient survival in breast cancer are discussed. In 1975, 88,700 new breast cancers were diagnosed in the US, corresponding to an incidence of 73.8/100,000 women. The incidence, which is low in younger women but which increases continuously throughout the life span, has risen consistently in Connecticut (and several metropolitan areas of the US) since 1935, the year when a cancer registry was started. The incidence in Connecticut was 56.3/100,000 between 1935 and 1939 and 79.6/100,000 between 1970 and 1972. The 5-yr survival rate has improved from 53% for patients diagnosed between 1940 and 1949 to 64% for patients diagnosed between 1965 and 1969.

Patients with localized disease have a more favorable prognosis than those with more extensive disease. The 5-yr survival rate for patients diagnosed between 1965 and 1969 was 84% for localized disease, 56% for regional disease, and 10% for disseminated disease. (no refs.)

**77-2904 Epidemiological Features of Choriocarcinoma.** (Eng.) Baltazar, J. C. (Dept. Epidemiology and Biostatistics, Inst. Public Health, Univ. Philippines, 625 Pedro Gil St., Ermita, Manila, Philippines.) *Bull WHO* 54(5): 523-532; 1977.

By means of hospital records and death certificates, 91 cases of choriocarcinoma were identified in the cities of Manila, Quezon, Pasay, and Caloocan in the Philippines during the 5 yr 1970-1974. The overall incidence was 17.4/100,000 live births. The other principal findings in this population-based study concerned maternal age, history of fetal wastage, and number of pregnancies. Very high incidence rates for choriocarcinoma were registered for mothers  $\geq 40$  yr and there was a slightly higher than av risk for women  $< 20$  yr of age. Risk increased with the number of fetal losses. The effect of number of pregnancies was evident only for very old and very young mothers. A case-control study was also conducted on 28 patients with choriocarcinoma and on 187 age-matched controls; for all of these women, the disease had been diagnosed (cases) or children had been born (controls) between 1970 and 1975. Compared with the control group, a higher proportion of the case group had a history of pulmonary tuberculosis (5/28 vs 7/186) and had previously used contraceptive pills (7/26 vs 10/185). Because of the low and unequal levels of cooperation among patients and controls, these findings need further confirmation. (31 refs.)

**77-2905 First-Trimester Exposure to Progestagen/Oestrogen and Congenital Malformations (Letter to Editor).** (Eng.) Goujard, J. (INSERM Groupe de Recherches Epidemiologiques sur la Mere et l'Enfant, 78110 le Vesinet, France) Rumeau-Rouquette, C. *Lancet* 1(8009): 482-483; 1977.

The effect of hormonal agents administered during the first trimester on teratologic defects was investigated. There was a significantly higher incidence of microcephalics and infants with transposition of the great vessels in children who had been exposed to the hormones. (4 refs.)

**77-2906 Hormone-dependent Neoplasms of the Canine Perianal Gland.** (Eng.) Hayes, H. M. (Environmental Epidemiology Branch, NCI, Bethesda, MD 20014) Wilson, G. P. *Cancer Res* 37(7): 2068-2071; 1977.

The epidemiological features of 472 dogs with microscopically confirmed neoplasms of the perianal gland are described.



These general characteristics suggest etiological factors similar to those responsible for hormone-related neoplasms in human beings. Perianal gland tumors appear to be androgen-dependent. Male dogs show a 5.6-fold increased risk compared with females: endogenous estrogens offer protection, and the use of estrogenic hormones is conventional therapy for the benign lesion. Both sexes of the cocker spaniel breed show an excessively high risk, suggesting that this dog family may be a model for genetic studies that could be relevant to familial aggregations of hormone-related tumors in men and women. Adrenocortical hormones may play a role in tumor development in female dogs. Research into alterations of the biochemical pathways of steroidogenesis in affected female dogs may provide clues to similar conditions in humans. (19 refs.)

- 77-2907 **Metabolic Epidemiology of Colon Cancer: Fecal Bile Acids and Cholesterol Metabolites of Patients with Colon Cancer, Adenomatous Polyps, Familial Polyposis or Ulcerative Colitis (Meeting Abstract).** (Eng.) Reddy, B. S. (American Health Foundation, Valhalla, NY) Weisburger, J. H.; Martin, C. W.; Hedges, A.; Wynder, E. L.; Lipkin, M. *Gastroenterology* 72(5/Part 2): 1117; 1977. (no refs.)

- 77-2908 **Dietary Influence on Colonic Cell Renewal.** (Eng.) Stragand, J. J. (Cancer Res. Unit, Div. Radiation Oncology, Clinical Radiation Therapy Res. Center, Allegheny General Hosp., 320 E. North Ave., Pittsburgh, PA 15212) Hagemann, R. F. *Am J Clin Nutr* 30(8): 913-923; 1977.

Previous studies have indicated that marked alterations in the proliferative activity of the murine intestinal epithelium can be induced through noninjurious fasting and refeeding. The response to refeeding is especially pronounced in the colon, where proliferative activity exceeds twice the control levels within 12 to 16 hr. The role of specific dietary components in the control of the colonic refeeding response and in normal colonic cell renewal was investigated. The results indicate that the colonic refeeding response after a 72-hr fast depends on the presence of sugar, amino acids and minerals, in the form of salts, in the refed diet. In the nonfasting animal, normal colonic cell renewal also requires dietary minerals. The absence of minerals in a maintenance diet results in a marked hypoproliferative state that can be reversed by reintroducing the minerals. The depressed proliferative rate observed with a salt-free diet is not the result of a caloric depletion, as shown by the maintenance of body wt throughout the 7-day experimental period. These findings suggest a potential role for specific dietary components in the control of colonic cell proliferation and maintenance of the steady-state cell renewal system, and they represent a tool by which the proliferative activity of the gut may be manipulated in HA/ICR female mice. (22 refs.)

- 77-2909 **Comparison of the Level of Mitotic Activity and Duration of Mitosis in Normal and Neoplastic Mouse Tissues During the 24-Hour Period.** (Eng.) Berezkin, M. V. (Lab. Chronobiology, Scientific-Res. Center, N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR) *Bull Exp Biol Med* 82(11): 1704-1706; 1976.

Experiments to determine diurnal changes in cell division in a carcinoma transplanted to the forestomach of male C3HA mice are reported. The mean diurnal mitotic activity of the tumor was two times higher than that of the epithelium of normal forestomachs. In contrast, demecolcine increased the accumulation of mitoses in the normal forestomach by 121.1% over a 24-hr period, compared with only 83.9% in the carcinoma. The larger number of tumor mitoses counted in the absence of demecolcine was explained by the fact that mean diurnal duration of tumor mitosis was 2.7 times greater than that of the normal forestomach. (10 refs.)

- 77-2910 **Kinetics of Induced Cell Proliferation at Steady-State Conditions of Cell Culture.** (Eng.) Yakovlev, A. Y. (Central Res. Inst. X-Ray Radiology, Ministry Public Health USSR, Pesochni-2, ul. Leningradskaya 70/4, 188646 Leningrad, USSR) Malinin, A. M.; Terskikh, V. V.; Makarova, G. F. *Cytobiologie* 14(2): 279-283; 1977.

The kinetics of cell proliferation was investigated in steady-state Chinese hamster fibroblastlike aneuploid cell cultures induced by medium change. A mathematical model demonstrated that quiescent cells, when induced to proliferate, enter the S period in two groups with a similar kinetic behavior. About 27% of the cells enter the first S period 8-16 hr after the induction of proliferation; 44% of the cells enter the first S period after 24 hr. The different behavior of these two groups of cells might be the result of their immediate environmental heterogeneity, particularly differences in monolayer density. In steady-state cultures, all cells induced to proliferate rapidly enter another mitotic cycle, which distinguishes them from cell populations such as regenerating liver. The mean prereplicative period was estimated to be 10 hr, which is longer than the mean duration of the G<sub>1</sub> period in the second mitotic cycle. The mean duration of the entire mitotic cycle was estimated to be 16 hr, which is consistent with the possibility of an early appearance of proliferating cells in the second mitosis. (10 refs.)

\* (Rev): 77-2402, 77-2405, 77-2406, 77-2407, 77-2408, 77-2409, 77-2410, 77-2411, 77-2416, 77-2417, 77-2418, 77-2423, 77-2425, 77-2426, 77-2428, 77-2429, 77-2430, 77-2438, 77-2443.

\* (Chem): 77-2503, 77-2504, 77-2536, 77-2547, 77-2552, 77-2555, 77-2563, 77-2568, 77-2573, 77-2579, 77-2589, 77-2591, 77-2594.

\* (Immun): 77-2810.

\* (Path): 77-2858, 77-2862, 77-2863, 77-2865.

## MISCELLANEOUS

7-2911 **Spontaneous Mutations in Ageing Human Cells: Studies Using a Herpesvirus Probe.** (Eng.) Fulfer, S. J. (Human Biology Dept., Chelsea Coll., Manresa Road, London SW3 6LX, England) *Mech Ageing Dev* 6(4): 271-282; 1977.

Old and young human fibroblasts were infected with three herpesvirus temperature-sensitive (ts) mutants (tsD, tsE, and tsG), and the reversion frequencies of the different mutants were measured on the virus harvested from these cells at the time of max cytopathic effect. The collected virus was plaque-assayed in BHK cells at 33 and 38.5 C. With tsE, the reversion rate in old cells was 100 times that in young cells. The tsD rates were similar in both types of cells. With tsG there was a fortyfold decrease in the reversion rate in the old cells. These differences are not due to different rates of virus production. The findings indicate that old cells alter the mutation rate of infecting herpesvirus in opposite directions, depending on the virus genotype, and that old cells are as proficient at virus production as young cells. It is suggested that these results are difficult to explain by any theory other than a derivation of the general error theory. Certain unknown factors limit the usefulness of viral probes in aging research and cast doubt on previous work of this type. (38 refs.)

7-2912 **Human Mammary Cancers in nu/nu-Mice. A Model for Testing in Research and Clinic.** (Eng.) Bastert, G. (Univ.-Frauenklinik, Theodor Sternkai 7, D-6000 Frankfurt/Main, W. Germany) Schmidt-Matthiesen, H.; Michel, R. T.; Fortmeyer, H. P.; Sturm, R.; Ford, D.; Gerner, R. *Klin Wochenschr* 55(2): 83-84; 1977.

Mammary tumors from 37 of 44 patients were successfully transplanted into nu/nu mice. Each of four rapidly growing tumors was sensitive to cyclophosphamide, adriamycin and methopterine when tested in vitro. A good correlation between original tumor and transplants was demonstrated, indicating the value of the nu/nu mouse as an animal model. (16 refs.)

7-2913 **Growth and Differentiation of a Primitive Nervous Cell Line after In Vivo Transplantation into Syngeneic Mice.** (Eng.) De Vitry, F. (Groupe de Neuroendocrinologie Cellulaire, Laboratoire de Physiologie Cellulaire, Coll. de France, 11 Place Marcelin Berthelot, 75231 Paris 05, France) *Nature* 267(5606): 48-50; 1977.

Evidence for a clonal line of a nerve cell precursor that after in vivo passage, yields distinct neuronal phenotypes is presented. The primitive nerve cell clone F7 (10<sup>7</sup> cells; a subclone of the C7 cell line isolated from a 14-day-old mouse hypothalamus embryo that synthesizes neurophysin and vasopressin) was injected sc into male A/J mice. Tumors arose after a latent period of 5 to 10 days, and they were excised and cultivated. Primary cultures were maintained for about 6 wk and then plated. The three distinct morphological types observed were separated and cloned. The first population of cells had the same chromosome number as the original F7 clone. Gomori's paraldehyde fuchsin and  $\beta$ -glucuronidase histochemical reactions were negative. The immunocytochemical detection of neurophysin was also negative. The second population was positive for the histochemical reactions and positive for neurophysin. The third population was a neuronal refractile-cell type closely resembling the C7 clone. In conclusion, the primitive nerve cell line F7, when injected sc into syngeneic mice, gives rise to tumors that contain not only the primitive cells but also a new type of differentiated cells. (9 refs.)

77-2914 **Properties of a Cell Line from Human Adenocarcinoma of the Rectum.** (Eng.) Watkins, J. F. (Dept. Medical Microbiology, Welsh Natl. Sch. Medicine, Heath Park, Cardiff, CFI 4XN, Wales) Sanger, C. *Br J Cancer* 35(6): 785-794; 1977.

A new, highly differentiated line of cells derived from an adenocarcinoma of the rectum (HT55) is described. It differs from other colorectal tumor lines in four main features: the use of UV-inactivated Sendai virus in its development (ie, to attach tumor cell clumps to plastic bottles); its release of RNA-containing material with a density of 1.5-1.16 g/ml; and its ability to stimulate bone production in athymic mice and the growth of CBA mouse embryo fibroblasts in vivo. The karyotype and growth-cycle characteristics of the line are described. (16 refs.)

77-2915 **The Growth of Leukemic Cells In Vitro.** (Eng.) Dicke, K. A. (Dept. Developmental Therapeutics, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) Spitzer, G.; McCredie, K. B. *Cancer Bull* 29(1): 10; 1977.

The course of acute leukemia and the diagnostic problems it presents are reviewed. Because normal cell proliferation



cannot be distinguished from leukemic cell proliferation in vitro, the use of culture techniques to detect early leukemia or relapse of leukemia during clinical remission has been limited. A method is described in which phytohemagglutinin stimulates the growth of leukemic, but not of normal cells. This technique allowed for early detection of leukemic cells in remission in four cases. (no refs.)

- 77-2916 Cytologie and Cytochemistry of Colony Cells in Soft Agar Gel Culture from Normal and Leukemic Bone Marrow.** (Eng.) Beckmann, H. (Molekularbiologisch-hamatologische Arbeitsgruppe, Hamburg, W. Germany) Neth, R.; Soltan, H.; Mertelsmann, R.; Winkler, K.; Hausmann, K.; Hellwege, H.; Skrandies, G. *Haematol Bluttransfus* 19: 21-32; 1976.

Cytological and cytochemical studies were performed on 1,026 colonies from 15 normal and 95 leukemic samples of bone marrow in order to investigate the effect of specific differentiation factors. No segmented neutrophils were observed in 180 colonies from 15 normal controls, which consisted mostly of monocytes and macrophages with pure eosinophil colonies rarely observed. In untreated acute myeloblastic leukemia (AML) or in AML in relapse, there was a significant reduction in monocyte/macrophage colonies as compared to acute lymphoblastic leukemia (ALL) as well as an increase in pure eosinophil colonies probably as a result of the reduced number of normal myeloid precursor cells during the active stages of AML. The relatively high percentage of pure eosinophil colonies seems to represent the differentiated stages of a residual population of normal stem cells. During remission, fewer pure eosinophil colonies were observed in both ALL and AML as compared to normal controls, while the sum of pure eosinophil colonies and mixed colonies with eosinophils was of the same order as in normal bone marrow. The most significant observation was the presence of plasma cells in 174 colonies and blast cells in 20 colonies among a total of 926 colonies derived from leukemic bone marrow. Immunofluorescence was used to demonstrate that the plasma cells produced immunoglobulins (Ig) in vitro. In colonies from five leukemic patients, labeling with specific antisera against IgG, IgA, and IgM heavy chains revealed that some plasma cells show positive immunofluorescence with all three antisera; other plasma cells show positive immunofluorescence with only one antiserum. The presence of plasma cells in colonies from leukemic patients and their absence in normal colonies cannot be explained so far. Colonies containing blast cells derived from leukemic bone marrow suggest the presence of factors inducing proliferation and in some cases even differentiation of blast cells under soft agar culture conditions. (30 refs.)

- 77-2917 Cellular Aggregation of Transformed Cells: Correlation with Growth in Soft Agar and**

**Tumorigenic Potential (Meeting Abstract).** (Eng.) Putman, D. L. (Microbiological Associates, Inc., NCI, NIH, Bethesda, MD 20014) Park, D. K.; Rhim, J. S.; Huebner, R. J.; Ting, R. C. *Proc Am Assoc Cancer Res* 18: 156; 1977. (no refs.)

- 77-2918 Cell Survival in the Aggregate Form: A Property of Transformed Epithelial Cells (Meeting Abstract).** (Eng.) Steuer, A. F. (Biotech Res. Labs, Bethesda MD 20852) Hentosh, P. M.; Ting, R. C. *Proc Am Assoc Cancer Res* 18: 157; 1977. (no refs.)

- 77-2919 Doubling Time and the NMR Properties of Water in Human Breast Cancer Cell Lines (Meeting Abstract).** (Eng.) Beall, P. T. (Baylor Coll. Medicine, Houston, TX 77030) Cailleau, R. M.; Hazlewood, C. F. *In Vitro* 13(3): 204; 1977. (no refs.)

- 77-2920 Influence of Serum on the Growth of Human Breast Carcinoma Cells In Vitro (Meeting Abstract).** (Eng.) Lasfargues, E. Y. (Inst. Medical Res., Camden, NJ 08103) Coutinho, W. G. *In Vitro* 13(3): 204; 1977. (no refs.)

- 77-2921 Differential Serum Requirement for Attachment by Normal and Transformed Cells In Vitro (Meeting Abstract).** (Eng.) Rajaraman, R. (Dept. Medicine, Faculty Medicine, Dalhousie Univ., Halifax, N.S., Canada) MacSween, J. M.; Fox, R. A. *In Vitro* 13(3): 205; 1977. (no refs.)

- 77-2922 Fully Characterized Cultured Cell Lines from Human Tumors (Meeting Abstract).** (Eng.) Fogh, J. (Walker Lab., Sloan-Kettering Inst. for Cancer Res., Rye, NY 10580) Fogh, J. M.; Fogh, H.; Loveless, J.; Milder, D.; Wright, W. *In Vitro* 13(3): 175; 1977. (no refs.)

- 77-2923 Factors in Isolation of Continuous Cell Lines from Small Cell Anaplastic Carcinoma of the Lung (Meeting Abstract).** (Eng.) Pettengill, O. S. (Dartmouth Medical Sch., Hanover, NH 03755) Sorenson, G. D.; Maurer, L. H. *In Vitro* 13(3): 176; 1977. (no refs.)

2924 **Matrix-perfusion Cultivation of Human Choriocarcinoma and Colon Adenocarcinoma Cells (Meeting Abstract).** (Eng.) Rutzky, L. P. (Northwestern Univ. Medical Sch., Chicago, IL 60611) Tomita, J. T.; Calen-M. A.; Kahan, B. D. *In Vitro* 13(3): 191; 1977. (no refs.)

2925 **An Assay for Cell Invasiveness (Meeting Abstract).** (Eng.) Tickle, C. (Dept. Biology as Applied to Medicine, Middlesex Hosp. Medical Sch., London W1P 6DB, England) Crawley, A.; Wolpert, L. *Br J Cancer* 35(2): 249; 1977. (no refs.)

2926 **Plasminogen-Activator-Mediated Quasimalignant Characteristics of 3T3 Cells: Morphology Cell Kinetics (Meeting Abstract).** (Eng.) Urquhart, C. (Marie Curie Memorial Foundation, Res. Dept., The Hart, Oxted, Surrey, England) Wright, E.; Whur, P.; Gorton, M.; Williams, D. C. *Br J Cancer* 35(2): 255; 1977. (no refs.)

2927 **Biochemical Characteristics Determining the Rate of Tumor Growth in the Organism.** (Eng.) Beev, V. N. (Div. Molecular Pharmacology, Scientific Inst. Biological Testing Chemical Compounds, Kupav-Moscow Oblast, USSR) *Biochemistry (NY)* 41(12): 1741-1747; 1977.

subcellular distribution and activity of hexokinase (HK) in normal and regenerating mouse liver were compared with those in solid and ascites hepatomas maintained in C3H/10T. In contrast to normal and regenerating liver, most of the HK in the hepatomas was bound to the mitochondrial membrane. The ratio of the bound HK (HK-b) to the total (HK-t) activity decreased with hepatoma growth. Malignant degeneration of the hepatocytes also led to a sharp decrease in their cytochrome oxidase (CO) activity. The data are in accord with Warburg's hypothesis, but a direct correlation was not observed between the malignancy of the hepatomas evaluated by their growth rate, and the biochemical parameters of the tumors. On the basis of Warburg's principle hypothesis, it is proposed that the energy metabolism of tumors can be evaluated by the activity and subcellular distribution of HK and the activity of CO in accordance with the following expression:  $(HK-t)^2 / HK-b \cdot CO + HK-b \cdot CO$ . A linear relationship exists between the energy metabolism of hepatomas and their growth rate. The application of these findings to transplanted Ehrlich ascites carcinoma is discussed. (34 refs.)

77-2928 **Effect of Cytochalasin B on Normal and Transformed Cultured Cells: Correlation Between Nucleation and Survival (Meeting Abstract).** (Eng.) Putzrath, R. K. (Temple Univ., Philadelphia, PA 19122) Brownstein, B. L. *In Vitro* 13(3): 166; 1977. (1 ref.)

77-2929 **Relation of Nucleo-Cytoplasmic Constants to the Optical Density of Cell Nuclei for States Characteristic of the Epithelial Field.** (Rus.) Gel'fandbein, Ia. A. (Riga, USSR) Kaplan, B. L.; Maerovich, I. M. *Eksperimental'naia Khirurgiya i Anestezia* (5): 72-76; 1976.

Various optical density constants for normal, precancerous and cancerous cell nuclei were found to have a direct functional relationship to the nucleocytoplasmic constants of the epithelial field. These constants may be diagnostically useful. (5 refs.)

77-2930 **Somatic Cell Hybrids Between Friend Erythroleukemia Cells and Mouse Hepatoma Cells.** (Eng.) Conscience, J. F. (Biology Dept., Kline Biology Tower, Yale Univ., New Haven, CT 06520) Ruddle, F. H.; Skoultschi, A.; Darlington, G. J. *Somatic Cell Genet* 3(2): 157-172; 1977.

Somatic cell hybrids were formed between bromodeoxyuridine-resistant murine hepatoma Hepa 1a cells, which synthesize and secrete serum albumin, transferrin, and  $\alpha$ -fetoprotein, and thioguanine-resistant Friend erythroleukemic parental cells, which synthesize globin messenger RNA (mRNA) and have carbonic anhydrase and acetylcholinesterase activity (these erythroid properties are stimulated in vitro by the addition of dimethyl sulfoxide). The hybrids were studied for expression of liver-specific and erythroid properties. Several independent clones were isolated by HAT selection and shown to be true hybrids by isozyme and chromosome analysis. All the clones displayed complete extinction of Hb and globin mRNA production and of the capacity to respond to dimethyl sulfoxide. Conversely, all the clones retained the liver-specific functions of albumin and transferrin secretion, although murine  $\alpha$ -fetoprotein appeared in only 2/9 clones tested. These data suggest that erythroid differentiation is actively inhibited by the hepatoma genome. This could indicate that albumin and transferrin expression is controlled in a different way from the steps leading to erythroid differentiation or that it is due to chromosome imbalance. (26 refs.)

77-2931 **Growth In Vitro of Tumour Cell x Fibroblast Hybrids in Which Malignancy Is Suppressed.** (Eng.) Straus, D. S. (Sir William Dunn Sch. Pathology, Univ.



Oxford, Oxford OX1 3RE, England) Jonasson, J.; Harris, H. *J Cell Sci* 25: 73-86; 1976.

Hybrid cells in which malignancy is suppressed were formed by the fusion of an immunoresistant variant of a Moloney leukemia virus-induced lymphoma with embryo fibroblasts and by the fusion of thioguanine-resistant clonal derivatives of a spontaneous melanoma with fibroblasts. The parental cells, the hybrids, and malignant segregants derived from the hybrids were analyzed for serum requirement, cloning efficiency in soft agarose, density-dependent growth inhibition, and secretion of plasminogen-activating enzyme. One malignant segregant from the lymphoma x fibroblast cross was gen-activating enzyme. One malignant segregant from the lymphoma x fibroblast cross was found by several criteria to have a more highly transformed phenotype than the hybrid from which it was derived. However, in the case of the melanoma x fibroblast crosses, none of the parameters examined could be correlated in a direct way with malignancy. (18 refs.)

**77-2932 Differential Effects of Mitochondrial Inhibitors on Normal and Tumorigenic Mouse Cells (Meeting Abstract).** (Eng.) Howell, N. (Sidney Farber Cancer Inst., Boston, MA 02115) Sager, R. *Fed Proc* 36(3): 337; 1977. (no refs.)

**77-2933 Critical Difference Between Normal and Cancer Cells, and the Possible Origin of the Hayflick Limit (Meeting Abstract).** (Eng.) Goldacre, R. J. (Chester Beatty Res. Inst., Fulham Road, London SW3 6JB, England) *Br J Cancer* 35(2): 247-248; 1977. (1 ref.)

**77-2934 Membrane Composition of Normal, Transformed, and Revertant Mouse Fibroblasts (Meeting Abstract).** (Eng.) Vessey, A. R. (Case Western Reserve Univ., Cleveland, OH 44106) *Diss Abstr Int [B]* 37(12/Part 1): 5986-5987; 1977. (no refs.)

**77-2935 Methionine Biosynthesis in Normal and Transformed Fibroblasts.** (Eng.) Kamely, D. (Dept. Molecular Hematology, Natl. Heart and Lung Inst., NIH, Bethesda, MD 20014) Weissbach, H.; Kerwar, S. S. *Arch Biochem Biophys* 179(1): 43-45; 1977.

The biosynthesis of methionine in simian virus 40 (SV40)-transformed human fibroblasts (strain GM-637) and their normal counterpart (strain GM-37) was studied. Normal fi-

broblasts were able to grow in a medium supplemented with methionine or homocysteine, but transformed fibroblasts required methionine for growth. When normal cells were exposed to <sup>3</sup>H-thymidine (1 μM) for 24 hr in a medium containing homocysteine and then assayed by autoradiography approx 60% of the cell nuclei were labeled. Under the same conditions, only 3% of the transformed cell nuclei exposed to <sup>3</sup>H-thymidine in a medium containing homocysteine were labeled. SV40 transformed and normal fibroblasts were assayed for B<sub>12</sub> transmethylese activity. Extracts derived from transformed fibroblasts were slightly less active in their ability to synthesize methionine compared to normal cells. The propyl B<sub>12</sub>/methyl B<sub>12</sub> ratio was 0.7-0.8, indicating that both normal and transformed cells grown in hydroxy B<sub>12</sub> contained predominantly the transmethylese holoenzyme. N<sup>5</sup>-<sup>10</sup>-Methylene tetrahydrofolate reductase was also required for methionine synthesis, because this enzyme was necessary for the synthesis of N<sup>5</sup>-methyltetrahydrofolate. Cells grown in medium containing methionine were assayed for their reductase content. Extracts derived from transformed fibroblasts were as active as normal fibroblasts in their ability to synthesize N<sup>5</sup>-methyltetrahydrofolate. The results indicate that the absolute growth requirement for methionine in the transformed cells does not necessarily involve a deficiency in enzymes related to the synthesis of methionine. (13 refs.)

**77-2936 Differences Between Rat Liver Epithelial and Fibroblast Cells in Sensitivity to 8-Azaguanine and Metabolism of Purines (Meeting Abstract).** (Eng.) Berman, J. J. (Naylor-Dana Inst., Valhalla, NY 10595) Tong, C.; Williams, G. M. *In Vitro* 13(3): 196; 1977. (no refs.)

**77-2937 Genetics of Regulation in Cultured Mammalian Cells.** (Eng.) Siciliano, M. J. (Dept. Biology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) Bordelon, M. R.; Humphrey, R. M. *Cancer Bull* 29(1): 17-19; 1977.

Somatic cell hybrids were used to study the regulation of gene expression in cultured mammalian cells. Two variant cell lines derived from Chinese hamster ovary cells were also established, and the regulation of enzyme activity was examined. It was suggested that alterations in gene expression can take place in response to a mutagen and that the isolation of cell lines with a variant at a single locus be used for further study. (8 refs.)

**77-2938 Developmental Genetics of Neural Tumors.** (Eng.) Knudson, A. G. (Inst. Cancer Res., Fox

Chase Cancer Center, Philadelphia, PA) Meadows, A. T. *Cancer Bull* 29(1): 9; 1977.

Two-mutation models for the inheritance of neural tumors are discussed, with either one mutation inherited and the other occurring in a somatic cell, or both mutations occurring in a somatic cell. Thus, different tumors could represent expressions of mutations at different single genes. (no refs.)

77-2939 Realization of Genetic Information Programming the Synthesis of Pepsinogen-Pepsin in the Mucous Membrane and Tumors of the Stomach in Man. (Eng.) Seitz, J. F. (N. N. Petrov Res. Inst. Oncology, USSR Ministry Health, 188 646 Leningrad, USSR) Kalinovsky, V. P. *Neoplasma* 23(5): 541-547; 1977.

Analytical and preparative electrophoresis in 2.5% polyacrylamide gels identified up to 19-20 fractions of separate RNA species and groups in malignant stomach tumors and in the mucous membrane of patients with stomach cancer, ulcer, and polyposis. <sup>14</sup>C-uridine incorporation into the nuclear and separate fractions of the cytoplasmic RNA was more intensive in the stomach tumors than in the mucous membrane of the stomach. Pepsinogen-pepsin was synthesized by the bound polysomes of normal mucous membrane but not by the polysomes of malignant stomach tumors. The 16S-17S fractions of messenger RNA, isolated from the cytoplasmic RNA of a pig stomach mucous membrane, were able to simulate pepsinogen-pepsin synthesis in vitro. (13 refs.)

77-2940 Synthesis of Messenger RNA-like Molecules in Isolated Myeloma Nuclei. (Eng.) Mory, Y. Y. Massachusetts Inst. Technology, Cambridge, MA 02139) Geffer, M. L. *Nucleic Acids Res* 4(6): 1739-1757; 1977.

Nuclei were isolated from the MOPC 315 mouse myeloma cell line grown in tissue culture under conditions that allow protein synthesis. Under these conditions, RNA synthesis was linear for at least 60 min, but most preparations supported linear synthesis for up to 3 hr. The rate of GTP incorporation was directly proportional to the number of nuclei added to the incubation mixture. When nuclei were incubated in the presence of cytoplasmic extract, there was a twofold enhancement in the rate of RNA synthesis. Sedimentation of RNA in sodium dodecyl sulfate and formamide was heterogeneous from < 10S to > 45S, resembling RNA synthesized in vivo. Some of the RNA has properties in keeping with those expected for messenger RNA (mRNA). Fifty percent of the RNA synthesis was inhibited by  $\alpha$ -amanitin, an inhibitor of RNA polymerase II that is responsible for the synthesis of mRNA. After an incubation of 2 hr, 10% of the newly synthesized RNA was found outside the nuclei; it sedimented with a broad distribution at 18S. A large fraction of the RNA

that was released from nuclei in vitro promoted the formation of polyribosomes and contained molecules that were polyadenylated and had 5' caps. (34 refs.)

77-2941 Diversity of mRNA Sequences in Normal and Regenerating Liver (Meeting Abstract). (Eng.) Fausto, N. (Brown Univ., Providence, RI 02912) Colbert, D. A.; Tedeschi, M. V. *Proc Am Assoc Cancer Res* 18: 133; 1977. (no refs.)

77-2942 Purification of RNA from Normal and Neoplastic Liver for Molecular Hybridization Studies with Nonreiterated DNA Sequences Using Potassium Iodide Equilibrium Density Gradients (Meeting Abstract). (Eng.) Garrett, C. T. (Univ. South Florida, Tampa, FL 33620) Gonzalez, F.; Caine, M.; Wiener, D. *Fed Proc* 36(3): 1078; 1977. (no refs.)

77-2943 The Ribosomes of *Agrobacterium tumefaciens*: Isolation, Purification and General Properties of the Ribosomes of the Tumorigenic Strain B6806 and the Non-tumorigenic Strain IIBNV6. (Eng.) Knopf, U. C. (Dept. Biological Chemistry, Univ. California, Davis, CA 95616) *Int J Biochem* 8(5): 403-411; 1977.

Ribosomes were isolated from *Agrobacterium tumefaciens* strains B6806 (tumorigenic) and IIBNV6 (nontumorigenic) and analyzed. The hydrodynamic properties of the ribosomes and their subunits resembled those of *Escherichia coli* ribosomes. Gel electrophoresis of RNA, isolated by extraction from ribosomes with detergent, demonstrated that the 23S ribosomal RNA of both B6806 and IIBNV6 contains a discontinuity. Fifty-eight ribosomal proteins from the 70S ribosomes were resolved by two-dimensional gel electrophoresis. The two-dimensional ribosomal-protein electropherograms of the tumorigenic and nontumorigenic strain were similar. The molecular wt distribution of the ribosomal-proteins of *A. tumefaciens* and *E. coli* was also similar. (32 refs.)

77-2944 Transcriptional Inhibition by a Fraction of Chicken Liver Nuclei (Meeting Abstract). (Eng.) Hardy, K. J. (Dept. Biochemistry, Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232) Kilianska, Z.; Chiu, J.; Hnilica, L. S. *Fed Proc* 36(3): 807; 1977. (no refs.)

77-2945 Establishment of Mouse Embryo Cells In Vitro: Relationship of DNA Synthesis, Senescence, and Malignant Transformation (Meeting Abstract). (Eng.)



Meek, R. L. (Univ. California, Santa Cruz, CA 95064) Bowman, P. D.; Daniel, C. W. *In Vitro* 13(3): 185-186; 1977. (no refs.)

77-2946 **Temporal Relationship Between DNA Synthesis and Growth Inhibition in Dexamethasone-treated Rat Glioma Monolayer Cultures (Meeting Abstract).** (Eng.) Grasso, R. J. (Coll. Medicine, Univ. Southern Florida, Tampa, FL 33612) Wodzinski, S. F.; Johnson, C. E. *In Vitro* 13(3): 190; 1977. (no refs.)

77-2947 **Interaction of DNA with Anti-cancer Drugs: Copper-Thiosemicarbazide System.** (Eng.) Pillai, C. K. (Dept. Inorganic and Physical Chemistry, Indian Inst. Science, Bangalore-560012, India) Nandi, U. S.; Levinson, W. *Bioinorg Chem* 7(2): 151-157; 1977.

The interaction of copper(II) bis-(thiosemicarbazide)sulfate (TSC-Cu) with calf thymus DNA was investigated using UV and infrared (IR) spectroscopy. The UV spectra suggested that the TSC-Cu mixture interacted with the bases of DNA. Further evidence for this interaction was obtained by measuring the difference spectra. The IR spectra also indicated that both DNA and TSC-Cu are present and that the DNA is bound to TSC-Cu. The IR spectrum showed two new bands when TSC-Cu was bound to DNA. Similar bands had been reported for a Cu(II)-DNA system by previous investigators, who suggested that these bands may be due to the binding of Cu(II) to cytosine and guanine sites of DNA. (20 refs.)

77-2948 **Simultaneous Fluorescence Analysis of Plasma Membrane and DNA in Individual Cells Before and After Transformation (Meeting Abstract).** (Eng.) Hawkes, S. P. (Lab. Chemical Biodynamics, Lawrence Radiation Lab., Univ. California, Berkeley, CA 94720) Bartholomew, J. C. *Br J Cancer* 35(2): 251; 1977. (1 ref.)

77-2949 **An Inhibitor of <sup>3</sup>H-Thymidine Incorporation into Hepatic DNA: Purification and Mode of Action (Meeting Abstract).** (Eng.) Sekas, G. (Inst. Pathology, Case Western Reserve Univ., Cleveland, OH 44106) Cook, R. T. *Fed Proc* 36(3): 1078; 1977. (no refs.)

77-2950 **Level and Activity of Nucleic Acids in the Peripheral Blood WBC of Patients Suffering from Leukemia and Anemia.** (Rus.) Beloshevskii, V. A. (Dept.

Hospital Therapy, Voronezh Medical Inst., Voronezh, USSR) *Probl Gematol Pereliv Krovi* 21(11): 24-26; 1976.

Nucleic acids were assessed quantitatively (by fluorescent microscopy) in the WBC of leukemia patients and controls. Leukemic WBC showed an increased concentration of RNA, but the content of DNA was practically the same as in controls. It was suggested that the bright fluorescence of the leukemic WBC was due to activation of the DNA molecule. (10 refs.)

77-2951 **Subcellular Distribution of Aminoacyl-tRNA Synthetases in Various Eukaryotic Cells (Meeting Abstract).** (Eng.) Ussery, M. A. (Clayton Foundation Biochemical Inst., Dept. Chemistry, Univ. Texas, Austin, TX 78712) Tanaka, W. K. *Fed Proc* 36(3): 705; 1977. (no refs.)

77-2952 **Studies on the Ribonucleotide Reductase Activity in Synchronized Mammalian Cells (Meeting Abstract).** (Eng.) Walsh, P. R. (Univ. Pittsburgh, Pittsburgh, PA 15213) *Fed Proc* 36(3): 885; 1977. (no refs.)

77-2953 **The  $\gamma$ -Glutamyl Transpeptidase of Normal Rat Breast and Breast Cancer: Purification, Characterization, and Comparison of Isozyme Composition (Meeting Abstract).** (Eng.) Jaken, S. (Univ. Michigan Medical Sch., Ann Arbor, MI 48109) Mason, M. *Fed Proc* 36(3): 825; 1977. (no refs.)

77-2954 **Release of  $\gamma$ -Glutamyl Transpeptidase-Like Activity by Degranulating Endoplasmic Reticulum of Normal Rat Liver (Meeting Abstract).** (Eng.) Lin, J. (Dept. Pathology, Univ. Toronto, Toronto, Ontario, Canada) Lee, G.; Sarma, D. S.; Farber, E. *Fed Proc* 36(3): 1078; 1977. (no refs.)

77-2955 **On The Mechanism of Glutamine Utilization of *E. Coli* GMP Synthetase (Meeting Abstract).** (Eng.) Zalkin, H. (Dept. Biochemistry, Purdue Univ., West Lafayette, IN 47907) Truitt, C.; Reinert, J. J. *Fed Proc* 36(3): 794; 1977. (no refs.)

77-2956 **Hormonal Regulation of Glutamine Synthetase and Ornithine Aminotransferase in Normal and Neoplastic Rat Tissue.** (Eng.) Wu, C. (Depts.

logical Chemistry and Internal Medicine, Univ. Michigan Medical Sch., Ann Arbor, MI 48104). In: *Control Mechanisms in Cancer*. Criss, W. E.; Ono, T.; Fine, J. R., eds. (New York: Raven Press): Progress in Cancer Research and Therapy Vol. 1, pp. 125-138; 1976.

the hormonal regulation of glutamine synthetase and ornithine aminotransferase in normal and neoplastic liver and that of the rat is discussed. Glutamine synthetase is a constitutive enzyme in the rat liver and is not responsive to glutamine injection or to dietary glutamine alteration. Cortisol and thyroxine increased the liver enzyme of suckling rats but had no effect on the enzyme in mature rats. The enzyme level increased approximately sevenfold in hepatoma 7800 when cortisol or thyroxine were applied. Thyroidectomy reduced the enzyme by half in the hepatoma. In adrenalectomized rats, the effects of cortisol and thyroxine on increasing glutamine synthetase were additive, suggesting that the two hormones act on the enzyme through separate mechanisms. The constitutive enzyme in normal liver acts like an adaptive enzyme in hepatomas. Three lines of Morris hepatomas were used to determine whether ornithine aminotransferase responded to triamcinolone. The enzyme was unresponsive in hepatomas 9633, 8999, and 66, as well as in the normal host rat. The activity of the enzyme was greatly increased in hepatoma 66. Ornithine aminotransferase elevation in female kidney by estradiol was age dependent; the increase was 10 fold in old adults and only 1.7 fold in young adults. Dactinomycin given simultaneously with estradiol completely blocked the increase of the enzyme. This suggests that de novo synthesis of certain RNA is essential to the estradiol effect. Diethylstilbestrol, 17 $\alpha$ -ethynyl-17 $\beta$ -estradiol-3-methyl ether, and hexestrol also elevated ornithine aminotransferase in the kidney. Progesterone and 17 $\alpha$ -estradiol did not affect the enzyme. Cyclic AMP also elevated ornithine aminotransferase, and the effect was both sex and age dependent. (39 refs.)

77-2957 **Expression of Ligandin and Glutathione S-Transferase Activities by Cells in Tissue Culture.** (Eng.) Smith, G. J. (Fels Res. Inst., Temple Univ. Sch. of Medicine, Philadelphia, PA 19140) Huebner, K.; Litwack, G. *Biochem Biophys Res Commun* 76(4): 1174-1180; 1977.

The wide distribution of glutathione S-transferase (GST) activity toward 1-chloro-2,4-dinitrobenzene and 1,2-dichloro-4-nitrobenzene in nontransformed, transformed, and hybrid cell lines was examined. A significant number of the cell lines contained proteins that were antigenically related to rat liver GST-B (ligandin). Transferase activity levels were lower in the in vitro cell lines than they were in vivo. The Chinese hamster fibroblast line A3, the rat hepatoma tissue culture HTC, and the rat-human hybrid line 235D demonstrated activity toward 1-chloro-2,4-dinitrobenzene relative to other cell lines. Human cell lines showed no detectable

activity toward 1,2-dichloro-4-nitrobenzene, but the rat-human hybrids 235D and 235H demonstrated a significant difference in transferase activity toward 1-chloro-2,4-dinitrobenzene. Stable cell lines may provide useful systems for studies of the interaction among carcinogens, steroids, and GST and of the fate of these proteins in cellular transformation. (29 refs.)

77-2958 **Studies of Neuraminidase and Sialyl Transferase in the Regenerating Liver of Rats.** (Rus.) Gabrielian, N. D. (M. M. Shemiakin Inst. Biological Chemistry, Acad. Sciences USSR, Moscow, USSR) Valiakina, T. I.; Komaleva, R. L.; Lapina, E. B.; Khorlin, A. Ia. *Vestn Akad Med Nauk SSSR* (3): 50-54; 1977.

Both soluble and membrane-bound neuraminidase were found in the regenerating rat liver with max levels of activity occurring at 18 and 24 hr after surgery, respectively. Sialyltransferase reached a max 24 hr after partial hepatectomy. These findings indicated an increase in sialyl metabolism in regenerating rat liver during the S period. (8 refs.)

77-2959 **A New Assay for Cell-bound Neuraminidase.** (Eng.) Petitou, M. (Lab. Biochimie Structurale, U.E.R. de Sciences Fondamentales et Appliquees, F-45045 Orleans Cedex, France) Rosenfeld, C.; Sinay, P. *Cancer Immunol Immunother* 2(2):135-137; 1977.

A new method for the detection of cell-bound *Vibrio cholerae* neuraminidase consists of testing enzyme activity with radiolabeled sialoglycoproteins on the surface of treated cells. Results with human lymphoblastoid cells show that the enzyme borne by one cell is able to cleave sialic acid from another cell. (20 refs.)

77-2960 **Effects of Adrenergic Agents on Ornithine Decarboxylase Activity in Hamster Brain Tumor Cells (Meeting Abstract).** (Eng.) Hsu, W. H. (Sch. Veterinary Medicine, Purdue Univ., West Lafayette, IN 47907) Coppoc, G. L. *Fed Proc* 36(3): 950; 1977. (no refs.)

77-2961 **Rhythmic Glycolytic and Respiratory Enzyme Patterns of Normal and Virally Transformed WI-38 Cells (Meeting Abstract).** (Eng.) Lewis, N. J. (Univ. Illinois Medical Center, Chicago, IL 60612) Rutzky, L. P.; Arsenis, C.; Pumper, R. W. *In Vitro* 13(3): 197; 1977. (no refs.)



77-2962 Isolation of Protease Inhibitor from Human Urine Using an Improved Chymotrypsin-Triazine-CL-Sepharose Resin (Meeting Abstract). (Eng.) Kessner, A. (New York Univ. Medical Center, New York, NY 10016) Hodgins, L. T.; Troll, W. *Fed Proc* 36(3): 893; 1977. (no refs.)

77-2963 Structural and Kinetic Alterations in Adenosine Deaminase Associated with the Differentiation of Rat Intestinal Cells. (Eng.) Trotta, P. P. (Lab. Cell Metabolism, Memorial Sloan-Kettering Cancer Center, New York, NY 10021) Balis, M. E. *Cancer Res* 37(7): 2297-2305; 1977.

Forms of adenosine deaminase (ADase) in rapidly dividing vs differentiated epithelial cells of rat intestinal mucosa were compared. Two variants with isoelectric points at pH approx 4.85 (ADase I) and 4.80 (ADase II) were resolved by preparative and analytical isoelectric focusing. These forms, which could also be partially resolved by molecular exclusion chromatography, displayed apparent molecular wt values of 37,000 and 33,500, respectively. The Km values of adenosine for the two forms were significantly different (0.07 mM for ADase I and 0.38 mM for ADase II). Small differences were also observed in relative substrate specificity, but the pH-activity profiles for the two forms were essentially identical, with broad maxima between pH 6.5 and 9.0. The intensity of ADase I observed in analytical isoelectric focusing increased dramatically with respect to ADase II as the epithelial cells differentiated. The specific adenosine deaminase activity also increased with cellular differentiation by > 30 times expressed per milligram DNA or > 6 times expressed per milligram protein. Cycloheximide sharply reduced this increase in specific activity. Cycloheximide also prevented the increase in the activity of ADase I that is normally associated with differentiation in rat intestines. The data imply the formation of a particular enzyme variant characteristic of the differentiated cell by a mechanism related to active protein synthesis. (47 refs.)

77-2964 Hexosaminidase in Human Colonic Cancer (Meeting Abstract). (Eng.) Pretlow, T. G. (Dept. Pathology, Univ. Alabama Medical Center, Birmingham, AL 35294) Marks, M. E.; Kimball, P. M. *Fed Proc* 36(3): 1075; 1977. (no refs.)

77-2965 Purification of Tyrosinase in Human Malignant Melanoma and its Dynamic Transition from the Particulate to Soluble and Desialylated Forms (Meeting Abstract). (Eng.) Nishioka, K. (The Univ. Texas System Cancer Center M.D. Anderson Hosp. Tumor Inst., Houston, TX 77030) *Fed Proc* 36(3): 825; 1977. (no refs.)

77-2966 Behavior of Acid Phosphatase in Human and Experimental Brain Tumors (Meeting Abstract). (Ger.) Rath, F. W. (Halle/Saale, E. Germany) Felicetti, D.; Janisch, W.; Schreiber, D. *Zentralbl Allg Pathol* 121(3): 277; 1977. (no refs.)

77-2967 Alkaline Phosphatase Activity in Human Bladder Tumor Cell Lines. (Eng.) Benham, F. (MRC Human Biological Genetics Unit, The Galton Lab., Univ. Coll. London, 4, Stephenson Way, London NW1 2HE, England) Cottell, D. C.; Franks, L. M.; Wilson, P. D. *J Histochem Cytochem* 25(4): 266-274; 1977.

The cellular localization and isoenzyme pattern of alkaline phosphatase (AP) were determined in five cell lines (T24, RT4, RT112, J82, EJ) derived from human bladder carcinomas and shown not to be HeLa cells. Only the RT112 cells (relatively well-differentiated) had high AP activity at confluency in the untreated control cultures. The activity of the RT4 cells (also well-differentiated) was higher than that of the T24, J82, and EJ cells (more anaplastic than RT112 and RT4 but still retaining ultrastructural features of human bladder tumor cells). The effect of prednisolone treatment on AP activity was minimal in three of the lines; in T24 and RT112 there was a two- to threefold stimulation. Electrophoretic separation of the isoenzymes showed that RT112 and RT4 cells had three heat-stable bands equivalent to placental AP and three slower bands of a modified placental type. Prednisolone increased only the former. In T24 cells, the enzyme resembled liver AP. Cytochemical studies confirmed the presence of cell surface-associated, extramembranous placental-type enzyme in RT112 cells. These findings suggest a possible correlation between morphologic and enzymatic differentiation in relation to the neoplastic state. (41 refs.)

77-2968 Alkaline Phosphatase Activity in Chronic Myelogenous Leukemia Cells in Cultures. (Jpn.) Chiyoda, S. (Div. Hemopoiesis, Inst. Hematology, Jichi Medical Sch., Minakawachi-machi, Tochigi, Japan) Miura, Y. *Acta Haematol Jpn* 40(2): 172-176; 1977.

Bone marrow cells from patients with chronic myelogenous leukemia (CML) and from normal volunteers were cultured by the soft agar and suspension culture method. The alkaline phosphatase (AP) activity was determined histochemically. In soft agar culture, bone marrow cells from the patients formed colonies of the same size as those from normal volunteers. Colonies containing AP-positive cells appeared on the fifth or sixth day of culture in both leukemic and normal bone marrow cells. The number of colonies containing positive cells and the number of positive cells per colony increased as the culture period was prolonged. AP-positive cells were not observed in colonies consisting of macrophages or eosinophils. There was no difference between leukemic and normal

one marrow cultures in the number of colonies containing positive cells or in the number of positive cells per colony. Almost all the metaphases from the colonies made from CML marrow cells showed the Philadelphia (Ph<sup>1</sup>) chromosome. In a suspension culture of marrow cells from one CML patient, the AP activity of the neutrophils increased on the second day of culture. In this case, 50%-80% of the cells at metaphase possessed Ph<sup>1</sup> chromosomes. Low neutrophil AP activity in CML suggests either a reduction in AP synthesis, an inhibitory mechanism in AP synthesis, or the absence of an activating process. The results suggest that in CML, hemopoietic progenitor cells can differentiate to neutrophils, showing normal levels of AP activity in culture. (14 refs.)

77-2969 **Enhanced Expression of Alkaline Phosphatase in Hybrids Between Neuroblastoma and Embryonal Carcinoma.** (Eng.) Bernstine, E. G. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Koyama, H.; Phrussi, B. *Somatic Cell Genet* 3(2): 217-225; 1977.

Expression of alkaline phosphatase (AP) activity was studied in three independent hybrid cell lines isolated from the fusion of clonal lines of embryonal carcinoma (OTT 6050 B) and neuroblastoma (N81TG2) and in a series of sublines derived from the original hybrid clones. In early hybrid generations, all hybrid lines demonstrated an enhancement of AP activity in the stationary phase of the growth cycle. This enhancement was two to eight times greater than the activity of the parent carcinoma cell line. Heat denaturation studies indicated that it is the carcinoma AP that is expressed in the hybrids. Segregation for very high levels of AP activity was observed among the subclones of one hybrid line. The specific activities of the segregants ranged from 0.1 to 133 U/mg. (15 refs.)

77-2970 **Alkaline Phosphatase and Nucleoside Phosphatase Activities in Primary and Secondary Mammary Carcinomas (Meeting Abstract).** (Ger.) Knezevic, M. (Zagreb, Yugoslavia) Knezevic-Krivak, S.; Rode, B. *Zentralbl Allg Pathol* 121(3): 288-300; 1977. (no refs.)

77-2971 **Hydrolysis of Nucleoside Triphosphate in Plasma Membranes of the Hepatocytes of Normal, Regenerating and Foetal Livers and in Cancer Cells of Hepatomas.** (Eng.) Filippova, N. A. (Dept. Pathological Anatomy and Human Tumors, Oncological Res. Center, Acad. Medical Sciences USSR, Moscow 115478, USSR) *Lia Histochem Cytochem (Krakow)* 14(4): 233-237; 1976.

Electron-histochemical studies were performed to determine the localization and activity of nucleoside triphosphatase (NTPase) in normal, regenerating, and embryonic mouse liv-

er, in rapidly and slowly growing mouse hepatomas, and in human hepatocellular tumors. The results show that changes in NTPase activity in the plasma membrane of cancer cells can proceed in different directions. Some cells exhibit high magnesium-dependent NTPase activity over the whole membrane surface, but others have a low activity that is present only in certain regions of the membranes. The degree of the changes in enzyme localization and activity is dependent on the level of tumor differentiation and its growth rate. However, these changes are not strictly typical of malignancy. (10 refs.)

77-2972 **Characterization of Nuclear Protein Kinase Activities from Rat Liver and Hepatoma 3924A (Meeting Abstract).** (Eng.) Glazer, R. I. (Dept. Pharmacology, Emory Univ., Atlanta, GA 30322) *Fed Proc* 36(3): 960; 1977. (no refs.)

77-2973 **Characteristics of a Specific Histone H4 Protein Kinase from a Murine Lymphosarcoma (Meeting Abstract).** (Eng.) Masaracchia, R. A. (Dept. Biological Chemistry, Univ. California, Sch. Medicine, Davis, CA 95616) Walsh, D. A. *Fed Proc* 36(3): 810; 1977. (no refs.)

77-2974 **Proteolytic Activity in Liver Cells from Mouse, Rat, Ehrlich Ascites Carcinoma Bearing Mouse and in Ehrlich Ascites Carcinoma Cells.** (Eng.) Tekavcic, E. (Inst. Oncology, 610 00 Ljubljana, Yugoslavia) Skrk, J.; Suhar, A.; Lebez, D. *Neoplasma* 23(5): 515-522; 1977.

The activity of intracellular proteinases in the liver of CBA mice bearing Ehrlich ascites carcinoma was compared with that in the liver of normal CBA mice and Wistar rats. The intracellular proteolytic activities of normal mouse and rat liver differed at pH 3.5, 6.0, and 7.5; the difference was especially significant in lysosome-mitochondrial liver fractions at pH 6.0, in which mouse liver was twice as active as rat liver. The activity in liver cells from tumor-bearing rats was not significantly different from normal at pH 3.5, but it was significantly depressed at pH 6.0 and 7.0. In the supernatant of tumor homogenates, the proteolytic activity was 0.110 E<sub>750</sub> millimicron/mg nitrogen (mμ/mg N) at pH 7.6 and 0.154 mμ/mg N at pH 3.5. No activity was detected at pH 6.0. The possibility that cancer cells contain lysosome proteinases not present in normal cells is discussed. (25 refs.)

77-2975 **Protein Composition of Nuclear 30-40S Ribonucleoprotein (RNP) Particles in Hepatoma (Meeting Abstract).** (Eng.) Patel, N. T. (Univ. Texas Medical



Branch, Galveston, TX 77550) Holoubek, V. *Fed Proc* 36(3): 807; 1977. (no refs.)

77-2976 **Fractionation of Nucleolar Proteins and RNA Polymerase I by Step-Salt Extraction (Meeting Abstract).** (Eng.) Rothblum, L. I. (Dept. Pharmacology, Baylor Coll. Medicine, Houston, TX 77030) Kunkle, H. M.; Ballal, N. R. Choi, Y. C.; Crane, P. M. *Fed Proc* 36(3): 786; 1977. (no refs.)

77-2977 **Synthesis of Stage-specific Proteins.** (Eng.) Wright, D. A. (Dept. Biology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) *Cancer Bull* 29(1): 11; 1977.

Study of the synthesis of proteins and their expression at different stages of amphibian embryonic development revealed new proteins made only during the late blastula and gastrula stages. It was determined that these new proteins are produced primarily by the ectodermal cells. The changes in protein synthesis correlated with the shift from maternal cytoplasmic to nuclear control of gene expression in the developing embryo. (no refs.)

77-2978 **Electron Transfer Properties of Melanin and Melanoproteins.** (Eng.) Menon, I. A. (Clinical Science Div., Section Dermatology, Univ. Toronto, Medical Sciences Building, Toronto, Ontario, M5S 1A 8, Canada) Gan, E. V.; Haberman, H. F. *Pigm Cell* 3: 69-81; 1976.

Studies of several types of melanin showed that they can be oxidizing and reducing agents in various oxidation-reduction systems. The results indicate that basic components of the proteins bound to melanin may be blocking the active sites of melanin involved in the oxidation of NADH. The electron-transfer properties of melanin may be important in its protective role against radiation and toxic free radicals. It is suggested that inactive melanoprotein, as it occurs in the cell, is converted to active melanin on dissociation or degradation of the protein by agents such as radiation. Melanins synthesized from dopamine, adrenalin, adrenochrome, and hydroquinone also have electron-transfer properties similar to 3,4-dihydroxyphenylalanine (dopa-melanin). These studies indicate that melanin may be reversibly oxidized by ferricyanide and reduced by NADH and that the oxidized and reduced forms of melanin may participate in the electron-transfer reactions of melanin. (21 refs.)

77-2979 **Cells Derived From a Mammary Carcinoma Produce Type IV Collagen In Vitro (Meeting Abstract).** (Eng.) Daniel, J. C. (Northwestern Univ. Medical

Sch., Chicago, IL 60611) Kuettner, K. E. *In Vitro* 13(3): 204; 1977. (no refs.)

77-2980 **Immunoelectron Microscopy of Albumin Synthesis in Rat Hepatoma Ascites Tumor Cells (Meeting Abstract).** (Eng.) Lin, C. (Dept. Pathology, Natl. Taiwan Hosp., Taipei, Taiwan, Republic of China) Chang, J. P. *Fed Proc* 36(3): 1066; 1977. (no refs.)

77-2981 **Local Inhibition of Centripetal Particle Transport Where LETS Protein Patterns Appear on 3T3 Cells.** (Eng.) Albrecht-Buehler, G. (Cold Spring Harbor Lab., P.O. Box 100, Cold Spring Harbor, NY 11724) Chen, L. B. *Nature* 266(5601): 454-456; 1977.

Various experimental procedures indicated that centripetal transport of particles attached to the surface of cells is inhibited by a surface network composed primarily of large external transformation-sensitive (LETS) protein. It is not known if LETS protein also inhibits cell motility. (23 refs.)

77-2982 **Cell Surface Changes Caused by Growth of B16 Melanoma Cells In Bromodeoxyuridine.** (Eng.) Evans, I. (Dept. Pharmacology and Toxicology, Univ. Rochester Sch. Medicine and Dentistry, Rochester, NY 14642) DiStefano, P.; Case, K. R.; Bosmann, H. B. *FEBS Lett* 78(1): 109-112; 1977.

Treatment of cultured B16 mouse melanoma cells with bromodeoxyuridine (BUdR) (3 µg/ml for 2 days) greatly increased the adhesivity of the cells without changing their electrophoretic mobility. However, treatment did change the partition ratio of B16 cells in aqueous two-phase dextran-polyethylene glycol, suggesting that basic alterations occurred on the cell surface. The possibility that incorporation of BUdR prevents the synthesis of membrane proteins required for cell differentiation and/or oncogenicity is discussed. (16 refs.)

77-2983 **Surface Membrane Glycoproteins of Cultured Human Cells from Colonic Tumors and Fetal Intestine (Meeting Abstract).** (Eng.) Tsao, D. (Veterans Admin. Hosp., Univ. California, San Francisco, CA) Kim, Y. S. *Gastroenterology* 72(5/Part 2): 1142; 1977. (no refs.)

77-2984 **The Effects of Cytidine Monophosphate on the Regeneration of Sialoproteins on the Surface of Cultured Lymphoma Cells.** (Eng.) Maca, R. D. (Dept. Medi-

cine, Div. Hematology-Oncology, Univ. Iowa, Coll. Medicine, Iowa City, IA 52242) Hakes, A. *Biochem Biophys Res Commun* 74(4): 1660-1666; 1977.

The regeneration of surface sialic acid on lymphoblastoid cells (Raji) in the presence of cytidine monophosphate (CMP) was studied. After the Raji cells were incubated with neuraminidase, approx 55% of the total cellular sialic acid was removed. When these cells were washed and incubated again with neuraminidase, only an additional 2.5% was removed. When 2 mM CMP was added to the regeneration medium, the amount of sialic acid regenerated was 0.728  $\mu\text{g}/10^7$  cells, a control value of 0.729  $\mu\text{g}/10^7$  cells. To determine whether the CMP-containing medium remained inhibitory to sialyl transferase throughout the 16-hr incubation period, the medium containing CMP was removed after incubation and tested for its inhibitory activity. This postincubation CMP medium inhibited surface sialyl transferase activity by 94.7% compared to control medium. CMP was ineffective in inhibiting the regeneration of surface sialoproteins. However, when cells were incubated with  $10^{-6}$  M emetine for the entire 16-hr incubation period, 56.2% of the surface sialoprotein was regenerated. Similarly, when the cells were incubated with  $10^{-4}$  M emetine for only 1 hr, washed, and then incubated in emetine-free medium for 16 hr, 57.1% was regenerated. The decrease in regeneration was not simply due to cell death, since the viability of the emetine-treated cells was 85%-90%, not different from the control cells. During the 16-hr incubation, the incorporation of  $^{14}\text{C}$ -leucine into trichloroacetic acid-insoluble material was 2% of the control value, indicating that protein synthesis was significantly inhibited during regeneration. Emetine was more effective in inhibiting protein synthesis than surface sialoprotein regeneration. The surface sialoprotein that was regenerated after the addition of emetine was synthesized intracellularly prior to the addition of emetine and needed only to migrate onto the cell surface during this regeneration. Surface sialoproteins may be largely synthesized intracellularly instead of being assembled on the cell surface by the surface-located transferase system. (15 refs.)

77-2985 **Depleted Surface Sialo-Peptide in Leukaemic Cells (Meeting Abstract).** (Eng.) Smyth, H. (Dept. Biochemistry, Univ. Coll., Belfield, Dublin 4, Ireland) O'Kennedy, R. *Br J Cancer* 35(2): 255-256; 1977. (3 refs.)

77-2986 **Phenomenon of Crystallization of Biopolymers in a Malignant Tumour.** (Eng.) Visokinskas, A. A. (Kaunas Medical Inst., Kaunas, USSR) *biophysics* 21(3): 445-452; 1976.

The structural arrangement of various polypeptide compounds in Ehrlich carcinoma was investigated. It was found

that malignant proliferation is accompanied by an aggregation of tightly packed spherical or cylindrical elementary formations. Normal crystalline structure is also reviewed. (45 refs.)

77-2987 **Isolation and Characterization of Glycoconjugates from B16 Mouse Melanoma Tumors (Meeting Abstract).** (Eng.) Sheik-Fareed, V. (M.S. Hershey Medical Center, Pennsylvania State Univ., Hershey, PA 17033) Bhavanandan, V. P. *Fed Proc* 36(3): 825; 1977. (no refs.)

77-2988 **Differential Expression of  $\beta$  Globin Genes in Cultured Mouse Erythroleukemia Cells (MELC) (Meeting Abstract).** (Eng.) Nudel, U. (Columbia Univ., New York, NY 10032) Salmon, J.; Terada, M.; Fibach, E.; Bank, A.; Rifkind, R. A.; Marks, P. A. *Fed Proc* 36(3): 887; 1977. (no refs.)

77-2989 **Actions of Plant Cytokinins on Dog Follicular Cells In Vitro.** (Eng.) Fernandez-Pol, J. A. (Lab. Molecular Biology, Room 4B18, Building 37, NCI, NIH, Bethesda, MD 20014) Hays, M. T.; Binette, J. P. *Exp Mol Pathol* 26(2): 251-259; 1977.

A study was made of the effects of zeatin [trans-6-(4-hydroxy-3-methylbut-2-enyl)aminopurine] and kinetin (6-furfurylaminopurine) on the structure, levels of intracellular cyclic AMP, and in vitro response to thyrotropin (TSH) of canine thyroid follicular cells. Upon treatment of the cells with 1 mM zeatin or 0.5 mM kinetin, the cells adopted a concave form at the apical border, which showed a thin belt-like area apparently composed of compacted microfilaments. These changes were not associated with changes in cyclic AMP levels, and they were unaffected by the addition of 15  $\mu\text{g}/\text{ml}$  of actinomycin D or 15  $\mu\text{g}/\text{ml}$  of cycloheximide. They were inhibited by 5  $\mu\text{g}/\text{ml}$  of vinblastine, 5  $\mu\text{g}/\text{ml}$  of vincristine, and 10  $\mu\text{g}/\text{ml}$  of cytochalasin b. The formation of pseudopods induced by 50 milliunits/ml TSH or by 1 mM 8-bromoadenosine-3',5'-cyclic monophosphoric acid was inhibited by zeatin and kinetin, but the cyclic AMP response to TSH was still intact. It is proposed that the N<sup>6</sup>-adenine derivatives, zeatin and kinetin, may induce their changes in cell morphology by acting directly or indirectly upon microfilaments. (25 refs.)

77-2990 **Epidermal Growth Factor Stimulates Prostaglandin Biosynthesis by Canine Kidney (MDCK) Cells.** (Eng.) Levine, L. (Dept. Biochemistry, Bran-



deis Univ., Waltham, MA 02154) Hassid, A. *Biochem Biophys Res Commun* 76(4): 1181-1187; 1977.

Stimulation of prostaglandin (PG) synthesis in canine kidney (MDCK) cells by epidermal growth factor (EGF) and by arachidonic acid and/or serum was evaluated. In the presence of serum or arachidonic acid, two- to threefold more PG were produced by the MDCK cells. These cells produced 3 to 10 times more  $\text{PGF}_2\alpha$  than  $\text{PGE}_2$ . EGF at  $10^{-9}$  M stimulated the release of radioactively labeled PG and arachidonic acid from radioactively labeled MDCK cells. EGF, at  $10^{-9}$  and  $10^{-10}$  M, stimulated PG production by MDCK cells but not that by rabbit aorta endothelial cells (CLO), transformed mouse fibroblasts (MC5-5), mouse fibroblasts (3T3), and human fibroblasts (D-550). Although the evidence is strong that EGF plays a role in cell growth and development, little is known about its biochemical mechanisms of action. Deacylation of phospholipids and/or triglycerides and conversion of some of this arachidonic acid into  $\text{PGE}_2$  and  $\text{PGF}_2\alpha$  seems to be an early reaction of MDCK cells to EGF. (26 refs.)

- 77-2991 **Comparison of Glucocorticoid Receptors in Cytosols of Normal Liver, Liver of Tumor-bearing Organism, and Zajdela Hepatoma.** (Eng.) Dmitrieva, L. V. (Lab. Endocrinology, Biology Faculty, M. V. Lomonosov State Univ., Moscow, USSR) Volchek, A. G.; Rozen, V. B.; Adler, V. V.; Shapot, V. S. *Biochemistry (NY)* 41(10, part 2): 1502-1508; 1977.

Cell receptors for dexamethasone (dex) and hydrocortisone were studied and compared in vitro in liver cells from intact animals, Zajdela ascites hepatoma cells, and liver cells from the host organism at different periods of tumor development. Male Wistar rats weighing 150-200 g were used in the experiments. The binding of  $^3\text{H}$ -dex by the cytosol of liver cells of adrenalectomized animals at 0°C was due to one class of specific receptor molecules with an association constant ( $K_s$ ) of  $6.4 \times 10^8 \text{ M}^{-1}$  and with the number of sites ( $n$ ) equal to  $6.0 \times 10^{-13}$  mole/mg of protein. Competition of various unlabeled hormones for the binding of  $^3\text{H}$ -D by the cell receptors of intact animal livers showed that the receptors have marked hormonal stereospecificity. A gradual decrease in  $n$  and a slight decrease in  $K_s$  were observed as the hepatoma grew. The  $K_s$  of intact animal liver was  $3.8 \times 10^8 \text{ M}^{-1}$  and  $n$  was  $4.87 \times 10^{-13}$  mole/mg of protein for dex. For hydrocortisone the  $K_s$  was  $0.57 \times 10^8 \text{ M}^{-1}$  and the  $n$  was  $4.06 \times 10^{-13}$  mole/mg protein. In a buffer of low ionic strength, the steroid receptor complexes of the cytosols of liver cells and ascites hepatoma sediment in the 3S-4S region. In a high-ionic-strength buffer they sediment in the 6S-7S region. As the Zajdela ascites hepatoma grows, the affinity of the receptors for dex and hydrocortisone changes and the number of binding sites decreases. (25 refs.)

- 77-2992 **Effect of Prolactin and Bromocriptine on Growth of Transplanted Hormone-dependent**

**Mouse Mammary Tumours.** (Eng.) Briand, P. (Fibiger Lab., Ndr. Frihavnsgrade 70, DK-2100, Copenhagen 0, Denmark) Thorpe, S. M.; Daehnfeldt, J. L. *Br J Cancer* 35(6): 816-821; 1977.

The effect of prolactin (50  $\mu\text{g}$  ip, 3x/day) and/or progesterone (5-10 mg/wk sc) plus estrone (0.5  $\mu\text{g}/\text{ml}$  in drinking water) on the growth of transplanted GR mammary tumors was studied in spayed syngeneic mice. Ovine prolactin alone supported the growth of hormone-dependent tumors, ie, those that grew progressively in estrogen-treated but not in untreated spayed mice. The growth of hormone-independent tumors was not stimulated. Bromocriptine (0.2 mg/day sc), a compound that inhibits the release of prolactin from the pituitary gland, inhibited the growth of hormone-dependent tumors but had no influence on hormone-independent tumors. The results demonstrated the involvement of prolactin in the growth of transplanted, hormone-dependent GR mouse mammary tumors. (30 refs.)

- 77-2993 **Role of Estrogen Receptor and Cyclic AMP Binding Proteins in the Growth Control of Hormone-Dependent Mammary Carcinoma (Meeting Abstract).** (Eng.) Bodwin, J. (NIH, Bethesda, MD 20014) Clair, T.; Cho-Chung, Y. S. *Proc Am Assoc Cancer Res* 18: 117; 1977. (no refs.)

- 77-2994 **Estrogen Receptor Characterization of the R3327 Transplantable Prostatic Adenocarcinoma (Meeting Abstract).** (Eng.) Markland, F. S. (Dept. Biochemistry, USC Sch. Medicine, Los Angeles, CA 90033) Lee, L. *Fed Proc* 36(3): 913; 1977. (no refs.)

- 77-2995 **Steroid Receptors in the Human Prostate Detection of Tissue-Specific Androgen Binding in Prostate Cancer.** (Eng.) Hawkins, E. F. (Service de Medecine Interne et Laboratoire d'Investigation Clinique, Institut Jules Bordet, Rue Heger-Bordet 1, 1000 Brussels, Belgium) Nijs, M.; Brassinne, C. *Clin Chim Acta* 75(2): 303-312; 1977.

An attempt was made to locate the tissue-specific binding receptors of  $5\alpha$ -androstane- $17\beta$ -ol-3-one ( $5\alpha$ -dihydrotestosterone;  $5\alpha$ -DHT) in cytosols prepared from 25 surgically obtained benign prostatic hypertrophy (BPH) samples and 3 tissue samples containing prostatic cancer cells to determine the role of sex hormones in prostate gland disease. Steroid-receptor complexes and ligand binding to serum sex hormone-binding globulin (SHBG) were distinguished by combination experiments using sucrose gradient ultracentrifugation and agar gel electrophoresis. Gradient analysis of a cytosol from a cervical lymph node (CLN) containing meta-

static prostate tissue revealed 8S and 4S forms of high-affinity  $5\alpha$ - $^3$ H-DHT binding. Upon electrophoresis of gradient fractions from these zones, anodally migrating steroid-receptor complexes were found only in the 8S peak; the 4S region contained radioligand bound to cathodally directed SHBG. In experiments with two BPH samples heavily invaded with prostate cancer cells, only a 4S peak of radioligand binding was detected. Its multicomponent nature was uncovered electrophoretically when, in addition to SHBG, saturable, androgen-binding molecules appeared anodally. Their incomplete resolution from SHBG on a gradient might have prevented their identification had this been the only method used. In contrast to the cancer-containing tissues, no saturable  $5\alpha$ - $^3$ H-DHT binding, other than that to SHBG, was detected in any of the BPH samples analyzed. Agar gel electrophoresis may be useful for further investigations into the multicomponent nature of androgen binding of prostatic tissue. (18 refs.)

**77-2996 Production of Carcinoembryonic Antigen by Human Prostate Epithelial Cells In Vitro.** (Eng.) Williams, R. D. (Dept. Urologic Surgery, Univ. Minnesota Health Sciences Center, Minneapolis, MN 55455) Bronson, D. L.; Elliott, A. Y.; Fraley, E. E. *J Natl Cancer Inst* 58(4): 1115-1116; 1977.

Sixteen human prostate epithelial cell cultures were found to produce either carcinoembryonic antigen (CEA) or CEA-like substances. This antigen could be used to identify these cells in culture. However, it is not yet known whether human prostatic epithelial CEA is identical to CEA from colorectal cancer. (13 refs.)

**77-2997 Restricted Lateral Diffusion of Concanavalin A Receptors of Different Malignant Cells of the Nervous System.** (Eng.) Zagayansky, Y. (Lab. Neurobiologie et Microscopie Quantitative, CHU Bobigny, Univ. Paris XIII, 93000 Bobigny, France) Benda, P.; Bisconte, J. C. *FEBS Lett* 77(2): 206-208; 1977.

Malignant cells originating from various types of neural cells were used to measure the lateral diffusion of concanavalin A (Con A) receptors. The diffusion constant for the Con A receptors of neuroblastoma cells having round or noncomplicated shapes was  $(1.5 \pm 0.5) \times 10^{-11} \text{ cm}^2 \text{ reciprocal second}^{-1}$ . For morphologically differentiated cells with different culture densities the lateral mobility was increased to  $(3.0 \pm 0.5) \times 10^{-11} \text{ cm}^2 \text{ s}^{-1}$ . Lateral diffusion of Con A receptors was slower in malignant cells than in their nonmalignant counterparts. (18 refs.)

**77-2998 Significance of the Bacterial Steroid Degradation for the Etiology of Large Bowel Cancer.**

**VII. Methodology of the Identification of Degradation Products of Bile Acids.** (Ger.) Slemrova, J. (Hygiene-Institut der Johannes Gutenberg-Universitat Mainz, Hochhaus am Augustusplatz, D-6500 Mainz 1, W. Germany) Edenharder, R. *Zentralbl Bakteriol [Orig B]* 164(3): 235-249; 1977.

The bacterial transformation of cholic, chenodeoxycholic, and deoxycholic acid were identified by thin-layer and gas chromatography and mass spectrometry. In thin-layer chromatography, the mean error of retention factors, characterized by the standard deviation, was too large to distinguish clearly among bile acid transformation products with similar chromatographic properties. The influence of relative air humidity, however, was decisive. This effect became apparent only after a certain threshold value was exceeded. A dependence of the retention factors on temperature and some mutual interferences between bile acids in mixtures were also demonstrated. However, experimental conditions can be chosen in a manner that minimizes the influence of these factors. The gas chromatographic identification of trimethylsilyl ethers of bile acid methyl esters was disturbed by methylation and silylation residues and the insufficient resolution power of the packed columns; the gas chromatographic analysis, however, led mostly to correct results. The mass spectrometric identification was the most reliable. The reference spectra of five silylated bile acid methyl esters (lithocholic, deoxycholic, cholic, apocholic, and  $3\alpha,12\alpha$ -dihydroxy-7-oxo-5-cholanoic acid) and one silylated bile acid trimethylsilyl ester are presented. Combined gas chromatography-mass spectrometry is well-suited to this type of study. (12 refs.)

**77-2999 Differences in Triglycerides Between Nephromas and Unaffected Cortex of Tumour-bearing Human Kidneys.** (Eng.) Geers, R. (Lab. for Gerontology, Dietetics and Nutrition Res., State Univ. Ghent, Ghent, Belgium) Matthys, F.; Verdonk, G.; Popelier, G. *Arch Int Physiol Biochim* 85(1): 170-171; 1977.

An analysis of the triglyceride content of nephromas and the unaffected cortex of tumor-bearing human kidneys indicated that fatty acid incorporation into both is from pools with the same fatty acid composition. However, unlike the tumor, the cortex shows specificity as to the incorporation of these acids. (3 refs.)

**77-3000 Carbohydrates Associated with Surface and Nuclear Membranes of Neoplastic Cells (Meeting Abstract).** (Eng.) Price, M. R. (Cancer Res. Campaign Lab., Univ. Nottingham, Univ. Park, Nottingham NG7 2RD, England) Stoddart, R. W. *Br J Cancer* 35(2): 250; 1977. (no refs.)





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# CARCINOGENESIS ABSTRACTS

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# CARCINOGENESIS ABSTRACTS

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## ABBREVIATIONS

**JOURNAL** names are abbreviated according to the *List of Journals Indexed in Index Medicus, Abbreviation Listing*. If the journal is not listed in this, abbreviations are derived from the *International List of Periodical Title Word Abbreviations*.

**LANGUAGE** of the article is indicated in parentheses after the title and is represented by a three-letter code. The source for these codes is *MARC Manuals Used by the Library of Congress*, pages 183-187.

**ABBREVIATIONS** used in abstracts:

A	angstrom(s)	mOsm	milliosmolar
ACTH	adrenocorticotrophic hormone	max	maximum
ADP	adenosine diphosphate	mEq	milliequivalent(s)
AMP	adenosine monophosphate	min	minute(s)
ATP	adenosine triphosphate	ml	milliliter(s)
approx	approximately	μl	microliter(s)
av	average	mm	millimeter(s)
BCG	bacillus Calmette-Guerin	mo	month(s)
bid	twice daily	mol wt	molecular weight
C	degree(s) centigrade	N	normal concentration
cal	calorie(s)	NAD	nicotinamide adenine dinucleotide
kcal	kilocalorie(s)	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
Ci	curie(s)	NADPH	reduced nicotinamide adenine dinucleotidephosphate
mCi	millicurie(s)	NCI	National Cancer Institute
μCi	microcurie(s)	NIH	National Institutes of Health
cm	centimeter(s)	PAS	periodic acid-Schiff
CNS	central nervous system	po	orally
cpm	counts per minute	ppb	parts per billion
DNA	deoxyribonucleic acid	ppm	parts per million
ED <sub>50</sub>	median effective dose	qid	four times daily
EDTA	ethylenediamine tetraacetic acid	qod	every other day
g	gram(s)	QO <sub>2</sub>	oxygen quotient
kg	kilogram(s)	R	roentgen
mg	milligram(s)	RBC	red blood cells (erythrocytes)
μg	microgram(s)	RNA	ribonucleic acid
Hb	hemoglobin	rpm	revolutions per minute
hr	hour(s)	sc	subcutaneous
ia	intra-arterial	sec	second(s)
id	intra-dermal	SGOT	serum glutamic-oxaloacetic transaminase
IgA	Immunoglobulin A	SGPT	serum glutamic-pyruvic transaminase
IgB	Immunoglobulin B	soln	solution
IgG	Immunoglobulin G	TCD	tissue culture dose
IgM	Immunoglobulin M	TCD <sub>50</sub>	median tissue culture dose
ILS	increased life span	tid	three times daily
im	intramuscular	UV	ultraviolet
ip	intra-peritoneal	WBC	white blood cells (leukocytes)
IU	International Unit(s)	wk	week(s)
iv	intravenous	wt	weight
Km	Michaelis constant	X	times
LD	lethal dose	yr	year(s)
LD <sub>50</sub>	median lethal dose		
M	molar		
μM	micromolar		

## REVIEW

- 77-3001 **The Mutagenicity of Hydrazine and Some of Its Derivatives.** (Eng.) Kimball, R. F. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) *Mutat Res* 39(2): 111-126; 1977.

The mutagenicity and carcinogenicity of hydrazine and some of its derivatives are reviewed. Hydrazine can react with DNA pyrimidines to saturate the 5,6- double bond, to form N<sup>4</sup>-aminocytosine, and to open up the pyrimidine ring with consequent loss of pyrimidines from DNA. Some of the substituted hydrazines react in much the same way, but others act as alkylating agents to alkylate purines. Hydrazine mutations are probably produced by direct mispairing at replication rather than by error-prone repair. Hydrazine produces mutations both in higher plants and in *Drosophila*. The mutations seem to be single-locus changes. No dominant lethals have been induced, even though mutations are produced in bacteria in the host-mediated assay. Some of the derivatives of hydrazine produce chromosomal aberrations and other chromosomal and nuclear effects. There is evidence that hydrazine, isoniazid, and several alkyl derivatives are carcinogenic in laboratory rodents. There is at yet no evidence for carcinogenicity in humans of isoniazid, the only compound for which there is appreciable epidemiological data. The mechanism for hydrazine carcinogenicity is not clear. (86 refs.)

- 77-3002 **Genetic Effects of Nitrous Acid.** (Eng.) Zimmermann, F. K. (Mykologie/Genetik, Technische Hochschule Darmstadt, Schnittpahnstrasse 10, 6100 Darmstadt, W. Germany) *Mutat Res* 39(2): 127-148; 1977.

Studies on the mutagenicity of nitrous acid are reviewed. At low pH, nitrous acid induces point mutations mainly by deamination of cytosine and adenine, but not guanine. More complex events, such as deletions and other chromosomal aberrations, may also be caused by DNA-DNA and DNA-protein crosslinks. Nitrous acid is mutagenic in bacteria, fungi, and some green plants. It is weakly mutagenic in *Drosophila*. Conclusive evidence for the induction of transmissible deletions by nitrous acid is only available for viruses and bacteria. In eukaryotes, there are only cytological data for structural aberrations induced by nitrous acid in chromosomes of the lilyflower, *Bellevia romana*. There is no evidence that nitrous acid induces mutation in the mitochondrial genes of an organism that responds very strongly to the nuclear genetic effects of this agent. Some primary lesions induced by nitrous acid are direct and do not require processing by cellular activities, but others must go through metabolic pathways before the final effect can be scored as a genetic

alteration. Some nitrous acid-induced damage can be repaired, but it is not known whether this applies to all types of primary lesions. Nitrous acid can also lead at low pH to the formation of nitrosamines, some of which, such as the dialkyl nitrosamines, are potent mutagens and carcinogens. Several studies using laboratory rodents have demonstrated the formation of carcinogenic and mutagenic nitrosamines after feeding with a combination of nitrites and secondary amines. (113 refs.)

- 77-3003 **A Review of Genetic Studies with Fluorescent Whitening Agents Using Bacteria, Fungi and Mammals.** (Eng.) Kilbey, B. J. (Dept. Genetics, Univ. Edinburgh, West Mains Road, Edinburgh EH9 3JN, Scotland) *Mutat Res* 39(2): 177-188; 1977.

Studies on the mutagenicity of fluorescent whitening agents (FWA) in microbial systems, *Salmonella typhimurium*, and mammalian systems are reviewed. The following six major structural types form the basis for all the important FWA's currently in use: stilbene derivatives, coumarin and carbostyryl compounds, 1,3-diphenyl-2-pyrazolines, naphthalimides, benzazolyl substitution products of ethylene, stilbene, thiophene, etc., and combined heteroaromatics. There is no evidence that any of the FWA's studied are mutagenic. Only in the case of one compound, disodium 4,4'-bis(2-sulfostyryl)biphenyl, could even the slightest indication of a positive response be obtained, and then only in the dominant lethal test using mice. Although this uniformly negative response may indicate nonmutagenicity of the agents, a more feasible explanation is that lack of penetration is contributing to the result. (18 refs.)

- 77-3004 **Mutagenicity and Metabolism of Vinyl Chloride and Related Compounds.** (Eng.) Bartsch, H. (International Agency Res. Cancer, Unit Chemical Carcinogenesis, 69008, Lyon, France) Malaveille, C.; Barbin, A.; Bresil, H.; Tomatis, L.; Montesano, R. *Environ Health Perspect* 17: 193-198; 1976.

Experimental data concerning the metabolism and mutagenicity of vinyl chloride (VC) and related compounds are reviewed. The data suggest that the biological effects of VC are related to its conversion by microsomal enzymes into chemically reactive alkylating agents that can bind covalently to various cellular macromolecules. The mutagenicity of VC to *Salmonella typhimurium* strain TA1530, which is reverted to his<sup>+</sup> by single base-pair substitutions, was increased 28-



fold after exposure to an atmosphere of 20% VC (volume/volume) in air. Hepatic microsomal mixed function oxidases from rats, mice, and humans were equally effective in transforming VC into alkylating agents in vitro. Two of the products of reaction with the microsomal enzyme system, chloroethylene oxide and 2-chloroacetaldehyde, demonstrated potent mutagenicity in microorganisms and Chinese hamster V79 cells. (31 refs.)

- 77-3005 Carcinogenic and Possible Mutagenic Effects of Stilboestrol in Offspring Exposed In Utero.** (Eng.) Bishun, N. P. (Res. Dept., Marie Curie Foundation, The Chart, Oxted, Surrey, England) Smith, N. S.; Williams, D. C.; Raven, R. W. *J Surg Oncol* 9(3): 293-300; 1977.

A literature review of the causal relationship between in utero exposure to stilbestrol and the later development of vaginal carcinoma in young women is presented. Many studies have shown the strength of this association. In one study of 66 females with clear-cell adenocarcinoma of the vagina, it was discovered that 49 of the patients had been exposed to stilbestrol in utero. The entire process appears to be triggered by hormonal changes at puberty, since a latent period of about 15-29 yr is required before the development of neoplasia. The critical period of exposure is around the eighth week of pregnancy, when the rudiments of the female lower genital tract are involved in organogenesis. The chemotherapeutic uses of stilbestrol and its effect on animals are also discussed. (27 refs.)

- 77-3006 Estrogen and Endometrial Carcinoma.** (Eng.) Knab, D. R. (Dept. Obstetrics and Gynecology, Uniformed Services Univ. Health Sciences, Bethesda, MD 20014) *Obstet Gynecol Surv* 32(5): 267-280; 1977.

The role of estrogens in the development of endometrial carcinoma (EC) is reviewed. Discussion is made of gonadal and extragonadal endogenous estrogen sources; the hypothalamic control of endogenous estrogens; exogenous estrogens; and receptor sites for estrogens. The following associations are considered pertinent to the role of estrogens in EC: (1) EC is associated with estrogen-secreting ovarian tumors and also with polycystic ovarian disease; (2) EC patients have an increased peripheral conversion of  $\delta 4$ -androstenedione to estrone; (3) the estrone/estradiol ratio is higher in postmenopausal women with corpus cancer than in those without; (4) EC is associated with hypothalamic "hyperactivity"; and (5) long-term administration of exogenous estrogen probably causes malignant changes in the endometrium. It appears likely that if there is adrenal androgen production of androstenedione associated with a normal to increased peripheral conversion to estrone a hormonal milieu is created

that can lead to EC in a susceptible patient if this hormone alteration persists. (187 refs.)

- 77-3007 Oestrogen Therapy and Endometrial Cancer** (Eng.) Anonymous (No affiliation given) *Br Med J* 2(6081): 209-210; 1977.

The number of studies linking hormone replacement therapy with endometrial cancer is now so great that this association cannot be ignored. The risk appears to increase with the duration of treatment. (22 refs.)

- 77-3008 Influence of Oral Contraceptives on Breast Diseases.** (Eng.) Fechner, R. E. (Dept. Pathology 1200 Moursund, Houston, TX 77030) *Cancer (Suppl)* 39(6): 2761-2771; 1977.

Three sources of information are available relative to the effect of oral contraceptive hormones on the breast: toxicity experiments in animals, histologic examinations of breast tissue from women taking hormones, and epidemiologic studies of oral contraceptive users. Animals treated with hormones at doses equivalent to human contraceptive levels have not developed mammary cancer at greater than expected frequency. Monkeys, whose reproductive cycle is similar to humans, have not developed cancer during long-term hormone administration except for a single animal in whom the disease was probably a chance occurrence. Histologic studies of human breast tissue have revealed no abnormalities attributable to hormones, with the rare exception of secretory changes indistinguishable from the normal lactating breast. Epidemiologic studies have usually shown a decreased frequency of benign disease and neither an increase nor a decrease in breast carcinoma. The one exception is a possible increased cancer risk in long-term oral contraceptive users with a history of previous surgery for benign breast disease. (78 refs.)

- 77-3009 Hepatic Tumors: Possible Relationship to Use of Oral Contraceptives.** (Eng.) Klatskin, G. (Dept. Medicine, Yale Univ., 333 Cedar St., New Haven, CT 06510) *Gastroenterology* 73(2): 386-394; 1977.

A review of the literature on the relationship between oral contraceptives and hepatic tumors is presented. It is suggested that mestranol may be the component with the greatest oncogenic potential. The presenting symptoms and appearance of the tumors are discussed. (107 refs.)

- 77-3010 First Human Cancer Link to Saccharin Found.** (Eng.) Anonymous (No affiliation given) *Chem Eng News* 55(26): 6-7; 1977.

An unpublished Canadian epidemiological study has indicated the first association of saccharin consumption with human cancer. It is stated that these new data show a risk factor at least twice that predicted by animal studies. (no refs.)

- 77-3011 Health Hazards from Cutting Fluids.** (Eng.) Kipling, M. D. (Employment Medical Advisory Service, Auchinleck House, Broad St., Birmingham B15 1DL, England) *Tribology Int* 10(1): 41-46; 1977.

Epithelioma and carcinoma of the scrotum are two types of cancer that can be traced to exposure to cutting fluids. The active carcinogens in the fluid (several ringed polycyclic hydrocarbons) should be removed from the oil. (no refs.)

- 77-3012 Can Synthetic Cutting Fluids Cause Cancer?** (Eng.) Evans, C. (No affiliation given) *Tribology Int* 10(1): 47; 1977.

Synthetic cutting fluids contain sodium nitrite and triethanolamine (which contains diethanolamine). The amine impurity can react with the nitrite to form the carcinogen N-nitrosodiethanolamine. It is not known whether the quantities inhaled or ingested could be carcinogenic. (6 refs.)

- 77-3013 Analysis of Atmospheric Carcinogens and Their Cofactors.** (Eng.) Sawicki, E. In: *Environmental Pollution and Carcinogenic Risks*. (Lyon: International Agency for Research on Cancer): IARC Scientific Publications No. 13, INSERM Symposia Series Vol. 52, pp. 297-354; 1976.

The problems of sampling and analysis of a variety of air pollutants, primarily those with carcinogenic, mutagenic, or cofactor activity, are discussed. Pollutants included benzo(a)pyrene (BaP), polynuclear aromatic hydrocarbons, aldehydes, alkylating agents, amines, chloromethyl ethers, epoxides, nitrosamines, halogenated compounds, nitrogen dioxide, nitrates, sulfate and sulfite. A variety of chemical and bioassay screening tests are also discussed, as well as screening test values and other useful screening tests for air pollutants. Experimental work on BaP is documented, and BaP concentrations ( $\mu\text{g}/1000 \text{ meters}^3 \text{ air}$ ) in urban atmospheres, highly polluted atmospheres and effluents, and in a large variety of environmental mixtures have been tabulated. (354 refs.)

- 77-3014 Toxicology and Drinking Water Contaminants.** (Eng.) Stokinger, H. E. (Nat'l. Inst. Occupational Safety and Health, Robert A. Taft Labs., Center Disease Control, U.S. Public Health Service, Cincinnati, OH) *J Am Water Work Assoc* 69(7): 399-402; 1977.

The concept of a threshold level of exposure to carcinogens and the extrapolation of carcinogenesis data from animal studies to man are discussed. It is contended that the threshold level (below which no biological response is observed) is a valid concept in carcinogenesis and that genetic inborn errors of metabolism may cause a shift in carcinogen thresholds. This genetic basis may underlie the susceptibility of certain exposed workers to the induction of liver hemangiomas by vinyl chloride (ie, 50 cases of hemangioma were reported among 25,000 heavily exposed workers). It is also stated that fallacies are involved in the extrapolation of data from high-dose animal experiments to normal low-dose human situations. This point is illustrated by a discussion of chloroform carcinogenesis. Chloroform levels currently found in drinking water are not believed to constitute a danger to health. (27 refs.)

- 77-3015 Feasibility of Monitoring Populations to Detect Environmental Carcinogens.** (Eng.) Muir, C. S.; MacLennan, R.; Waterhouse, J. A.; Magnus, K. In: *Environmental Pollution and Carcinogenic Risks*. (Lyon: International Agency for Res. on Cancer) IARC Scientific Publications No. 13, INSERM Symposia Series, vol. 52, pp. 279-294; 1976.

Population monitoring to detect the entry of a carcinogen into the environment is considered. Two approaches to cancer monitoring are described: passive or routine monitoring (also called monitoring without a hypothesis) and active or exposure-oriented monitoring (monitoring with a hypothesis). Passive monitoring deals with the whole population and uses mortality, morbidity, geographic data, and time factors. Sizeable populations are necessary in order to give statistical sensitivity, but even so a rise in a common cancer in a small group could still remain undetected. It is more successful to detect a change in a rare or unusual form of cancer, although it should be confirmed by histologic diagnosis as well as site incidence. Once a rise in incidence or mortality is detected, there still remains the identification of the carcinogenic agent; this can be complicated by the long and variable latency of some cancers as well as the possibility of more than one carcinogen causing the same result. Active or exposure-oriented monitoring is considered most useful for defined groups and exposures. Such monitoring, to be successful, demands not only an expensive data collection by employers, industry, and authorities, but also the cooperation of the public in allowing their personal details to be collected. Two tables, one comparing the use of mortality and morbidity data for passive monitoring and the other summarizing both methods, are included. (30 refs.)



- 77-3016 **Estimation of Risks Due to Environmental Carcinogenesis.** (Eng.) Cranmer, M. F. (Natl. Center Toxicological Res., Jefferson, AR 72079) *Med Pediatr Oncol* 3(2): 169-198; 1977.

The inexactness of current toxicological, epidemiological, and mathematical models for estimating risk due to exposures to DDT, aflatoxin B<sub>1</sub>, diethylstilbestrol, and benzidine is exemplified, and the impact of different laws and regulations for the control of these agents is discussed. The three major control strategies for the regulation of toxic substances, including carcinogens, are: (1) the all-or-none approach, eg, the Delaney Clause of the Food, Drug, and Cosmetic Act; (2) the use of safety factors; and (3) statistical extensions beyond the experimentally observable range (mathematical extrapolation models). The main deficiencies in past studies of carcinogenicity concerned experimental dosing and definition of end points. The development of a methodology for adequate evaluation of carcinogenic risk involves two major approaches: (1) establishment of a carcinogen dose-response relationship using various end points, such as tumor prevalence, time to tumor and life shortening, and (2) development of a methodology and concepts that will permit extrapolation of the results to man. (45 refs.)

- 77-3017 **Cocarcinogens as Environmental Hazards.** (Eng.) Scribner, J. D. (Pacific Northwest Res. Foundation, 1102 Columbia St., Seattle, WA 98104) *Med Pediatr Oncol* 3(2): 151-157; 1977.

Experiments on the cocarcinogenicity of various chemicals are reviewed, and the regulation of these agents is discussed. The distinction is made between a cancer initiator and promoter. In experiments on the two-step induction of skin tumors in mice, the effect of initiators was found to be irreversible and additive, whereas the effect of promoters depended on giving sufficiently large doses sufficiently close together. For regulatory purposes, the definition of a carcinogen should be restricted to any substance that induces a tumor type not usually observed. The current definition used by the Environmental Protection Agency and the Health, Education, and Welfare Toxicology Subcommittee for Carcinogen Standards is any substance that induces an increased incidence of a tumor type normally seen or causes such tumors to appear at an earlier time than would otherwise be expected. However, this is a definition of a cocarcinogen, not a carcinogen. Known human carcinogens should be removed from circulation if possible, or reduced to the lowest possible exposure if not. Experimental cocarcinogens, however, should be considered on a case-by-case basis, without any attempt to apply all available technology to achieve minimal exposure. (13 refs.)

- 77-3018 **Application of the Results of Carcinogen Bioassays to Man.** (Eng.) Shubik, P.; Clayson, D. B.

*In: Environmental Pollution and Carcinogenic Risks.* (Lyon: International Agency for Research on Cancer): IARC Scientific Publications No. 13, INSERM Symposia Series Vol. 5, pp. 241-252; 1976.

The problems of experimental design and interpretation of animal tests for the carcinogenic activity of chemicals in man are discussed. The difficulties encountered include the shorter life span of animals; the impurities and chemical intoxication that may result from high test doses; the high cost of bioassays, especially for two-generation tests; the difference in metabolism in each species; and the inability to quantify dose-response relationships. To illustrate that animal testing is, at times, an inexact model for human disease, studies on diethylstilbestrol, chloropenathane, estrogen, and progesterone are discussed. (27 refs.)

- 77-3019 **Scientific Bases of Environmental Carcinogenesis and Cancer Prevention: Developing an Interdisciplinary Science and Facing its Ethical Implications** (Eng.) Saffiotti, U. (Experimental Pathology Branch, Carcinogenesis Program, Div. Cancer Cause and Prevention NCI, Bethesda, MD 20014) *J Toxicol Environ Health* 2(6): 1435-1447; 1977.

The characteristic toxic event in carcinogenesis alters the regulatory mechanism of the target cells so that these proliferate and give rise to a progeny of permanently altered cells. Once this transformed population exhibits marked competitive growth advantage over the adjacent tissue, a clinically detectable tumor results, which will then grow and spread at a rate independent of the intensity of the original toxic event. (16 refs.)

- 77-3020 **Effect of Changed Working Techniques on Asbestos Dust Levels in the Working Environment.** (Eng.) Cross, A. A. *In: Environmental Pollution and Carcinogenic Risks.* (Lyon: International Agency for Research on Cancer): IARC Scientific Publications No. 13, INSERM Symposia Series, Vol. 52, pp. 121-126; 1976.

The progress made in occupational protection against asbestos fibers is discussed. Since asbestos has a fibrous structure, specialized dust monitoring equipment is needed. Controlling asbestos fibers in the demolition of old plants or stripping of old insulation has been achieved by thorough wetting out of the material together with strict standards of hygiene. Working with asbestos material away from factory facilities has previously been a problem; but new equipment involving dust extraction, vacuum equipment, and special hoods has enabled site cutting, drilling, routing, and sanding of asbestos to be carried out in safety. A program of educating employees in the correct use and maintenance of protective clothing and equipment has been promoted by the Environmental Control Committee. (no refs.)

**77-3021 Public Health Hazards from Electricity-producing Plants.** (Eng.) Neyman, J. (Statistical Lab., Univ. California, Berkeley, CA 94720) *Science* 195(4280): 754-758; 1977.

From previous studies on radiation effects, there is no way of estimating the number of cancer cases that could result from a nuclear power plant in a given neighborhood. The experience with Hiroshima and Nagasaki, and the results of animal experiments, however, indicate that further investigations are necessary. Epidemiological studies including many localities and types of pollutants are recommended. (18 refs.)

**77-3022 Low-Level Radiation: Predicting the Effects (Letter to Editor).** (Eng.) Wolfe, B. (Fuel Recovery and Irradiation Products Dept., General Electric Co., San Jose, CA 95125) *Science* 196(4297): 1387-1389; 1977.

Previous articles espousing the use of the linear theory to predict cancer incidence from radiation exposure are reviewed, and the various arguments are refuted. When the linear theory was applied to 1,250 children exposed to atomic bomb radiation in Japan, an extra 18 deaths were predicted; in fact, none were observed. Another report concerns cancer incidence as a function of the bone dose of radiation received by workers on radium watch dials. In a population of 600, the 500 who received less than a cumulative bone dose of 1,000 rads were free of cancers. Of the 100 people who received 1,000-50,000 rads, the mean cancer incidence was 28%, independent of dosage. A good fit of the linear theory is not possible here. An inverse logarithmic relationship between dose and latent period was seen in the radium workers, in beagles inhaling plutonium, in uranium miners, in animals exposed to x-rays, and in Japanese atomic bomb survivors. An increase of latent period with decreased radiation exposure implies a practical threshold in which the latent period is longer than the life span. This theory is at variance with the linear theory. The linear theory predicts that a background radiation exposure of 170 millirems per year would cause a 2% increase in the cancer death rate. A recent study, however, showed a 20% decrease in cancer mortality at this exposure. (13 refs.)

**77-3023 Guidelines for Detection, Diagnosis, Treatment, and Follow-up of Radiation Related Thyroid Cancers.** (Eng.) Flowers, W. M. (Dept. Radiology, Univ. Mississippi Medical Center, 2500 N. State St., Jackson, MS 39216) Sanders, J. A. *J Miss State Med Assoc* 18(7): 170-172; 1977.

Individuals who received therapeutic doses of x- or  $\gamma$ -radiation to the head, neck, and upper thorax for various nonmalignant conditions during childhood are at increased risk of developing thyroid cancer. Without previous history

of irradiation, only one case of thyroid cancer in 27,000 is expected. Of 100 asymptomatic individuals with a history of irradiation to the head and neck, 26% had palpable abnormalities, and 7 of 15 patients who were surgically explored had malignant tumors. In another survey, 1,056 previously irradiated patients were studied; of the 182 patients who were surgically explored and analyzed, 33% had carcinomas. (9 refs.)

**77-3024 Molecular Biology of the Response of Cells to Radiation and to Radiomimetic Chemicals.** (Eng.) Strauss, B. S. (Dept. Microbiology, Univ. Chicago, 920 E. 58th St., Chicago, IL 60637) *Cancer (Suppl)* 40(1): 471-480; 1977.

Radiation and radiomimetic chemicals can be carcinostatic, carcinogenic, and mutagenic. In all cases, the critical reaction is with the cellular DNA, in which both ionizing radiation and radiomimetic chemicals produce a variety of adducts and changes. Human cells respond to these lesions in several ways. Some adducts are ignored. Others are recognized as different by the excision repair mechanism and are cut out of the DNA. Other adducts may be bypassed by special post-replication repair mechanisms so that viable daughter cells still containing altered DNA are produced. Unrepaired lesions may lead to chromosome aberrations and cell death. Since only viable cells can produce tumors, postreplication repair is critical to the initial events in carcinogenesis. Lesions converted to DNA strand breaks, on the other hand, lead to cell death. Knowledge of the changes produced in DNA and understanding of the different cellular responses possible should permit prediction of the relative tumorigenic and tumorstatic properties of compounds. (43 refs.)

**77-3025 Radiation Injury: Some Aspects of the Oncogenic Effects.** (Eng.) Fry, R. J. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439) Ainsworth, E. J. *Fed Proc* 36(5): 1703-1707; 1977.

The principles of radiation injury are reviewed briefly. The experience gained from radiation studies should be instructive with regard to the more complex task of assessing risk from chemical carcinogens. Exposure levels and radiation injury, dose-response relationships and risk estimates, and factors that influence risk estimates and life shortening are discussed. (37 refs.)

**77-3026 RNA Tumour Viruses.** (Eng.) Weiss, R. (Imperial Cancer Res. Fund Labs., London, England) *Trends Biochem Sci* 2(7): N154-156; 1977.



Nondefective, strongly transforming Rous sarcoma virus (RSV) strains have been used for genetic analysis of the transforming function of RNA tumor viruses. Experiments using a specific DNA probe complementary to the *src* transformation gene of RSV and isolated transformation-defective variants have been conducted to study the *src* gene. This gene may exert its transforming effect by coding for a protein. The resultant oncoprotein possibly acts at the cell surface rather than in the nucleus. Although no gene products have been isolated thus far, evidence suggests that they may be functional analogs of specific growth factors. This is adduced from the observation that cells transformed by murine sarcoma virus lack receptors for epidermal growth factors. (24 refs.)

- 77-3027 **European Tumour Virology 1976.** (Eng.) Vaheiri, A. (Dept. Virology, Univ. Helsinki, Haartmaninkatu 3, SF-00290 Helsinki 29, Finland) *Med Biol* 55(1): 15-20; 1977.

Information on research in European viral oncology laboratories that was presented during a 1976 meeting is summarized. Both simian virus 40 (SV40) and polyoma viruses have only three genes, an early gene coding for the early RNA transcript and T antigen and two genes coding for the late messenger RNA (mRNA) transcripts. T antigen is the "transforming protein," but its mechanism of action is not understood. SV40-transformed cells also express U antigen and tumor-specific transplantation antigen (TSTA), both of which may contain portions of the T-antigen amino acid sequence. A crude map of the herpesvirus genome is beginning to emerge. The different herpesvirus DNA's all appear to contain a long  $70 \times 10^6$  to  $75 \times 10^6$ -dalton region of non-repeated nucleotide sequences (fundamental sequences). "Accessory" sequences are present in mammalian herpesviruses. Retrovirus components (reverse transcriptase, high-molecular-wt RNA) have been associated with human mycosis fungoides tumors; a lymphoid cell line established from one of these tumors produced infectious virus with typical retrovirus characteristics. Milk from some women also possesses reverse transcriptase activity, as well as particles similar to murine mammary tumor virus and the Mason Pfizer monkey virus. In investigations for antiviral antibodies, sera from patients with a variety of neoplastic diseases have so far shown no unusual antibody titers. (no refs.)

- 77-3028 **Epstein-Barr Virus, Infectious Mononucleosis, Burkitt's Lymphoma and Nasopharyngeal Carcinoma.** (Eng.) Klein, G. (Dept. Tumor Biology, Karolinska Inst., Stockholm, Sweden) *Isr J Med Sci* 13(7): 716-724; 1977.

The association of Epstein-Barr virus (EBV) with infectious mononucleosis, Burkitt's lymphoma (BL), and nasopharyn-

geal carcinoma (NPC) is reviewed. African BL may be regarded as the neoplastic proliferation of an EBV-genome-carrying clone of B lymphocytes in 97% of cases. The rare cases of BL occurring outside the highly endemic areas of Africa do not, as a rule, show association with EBV, either by serology or EBV genome tests. In contrast to BL, there appears to be no major geographical variation in the association of EBV with NPC. However, there is a striking histological restriction: only poorly differentiated or anaplastic NPC tumors have been found to carry the EBV genome. The possibility of an etiological relationship between EBV and BL and NPC is discussed. (93 refs.)

- 77-3029 **A Status Report: Human Prostatic Carcinoma, with Emphasis on Potential for Viral Etiology.** (Eng.) Ziegel, R. F. (Dept. Medical Viral Oncology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263) Arya, S. K.; Horoszewicz, J. S.; Carter, W. A. *Oncology* 34(1): 29-44; 1977.

Data are reviewed from the following sources relevant to the possibility of human prostatic cancer having a viral etiology: epidemiology, clinical studies, morphology, pathology, enzymology, immunology, endocrinology, model animal systems, in vitro systems and viral investigations. A fundamental barrier to more definitive studies is the lack of long term cultures of normal and pathological prostate epithelium from men of all ages. (282 refs.)

- 77-3030 **Introduction: Cause and Prevention of Bladder Cancer.** (Eng.) Rapp, F. (Dept. Microbiology, Milton S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA 17033) *Cancer Res* 37(8/part 2): 2937-2938; 1977.

Evidence is increasing that either endogenous or exogenous viruses may cause cancer in man. Carcinogens in the occupational environment may activate latent viruses, such as human herpesviruses and oncornaviruses. (10 refs.)

- 77-3031 **Normal Immune Response in Man and Its Reaction to Malignant Disease.** (Eng.) Harris, J. E. In: *The Immunology of Malignant Disease*. (St. Louis: C. V. Mosby Co.) 2nd ed., pp. 1-92; 1977.

Cellular events in the normal immune response are broadly outlined, and their relation to human malignancy is consid-

ered. Evidence is presented that supports the theory that antibody production (humoral immunity) and delayed hypersensitivity (cell-mediated immunity) are basically different immune processes. Other immunologic reactions, such as immunologic tolerance, immune deviation, immunologic enhancement, and allogeneic inhibition, may also be involved in tumor growth or rejection. The immune function in cancer patients may be affected by factors other than the disease itself; ie, cytotoxic drugs, radiotherapy, and postoperative immunodepression. The in vivo and in vitro parameters of both cell-mediated and humoral immune function have been investigated in clinical studies. The extent to which these host defense mechanisms are successful influences tumor spread, response to treatment, and ultimate prognosis. In clinical studies, the stage of disease may determine whether evidence of host reactivity can be demonstrated. There is convincing evidence for human tumor antigens and host reactivity to them in several major tumor types, including malignant melanoma, Burkitt's lymphoma, leukemia, sarcoma, and in genitourinary, breast, brain, skin, lung, and colon cancer. Carcinoembryonic antigens (carcinoembryonic antigen,  $\alpha$ -fetoprotein) are of growing importance in tumor immunology; they are identified by immunologic techniques but may not be themselves immunogenic in the autochthonous host. Immune escape mechanisms allow tumor cells to escape either or both detection and elimination by immune responses. (975 refs.)

**77-3032 Immune Deficiency States Associated with Human Malignant Disease.** (Eng.) Harris, J. E. In: *The Immunology of Malignant Disease*. (St. Louis: C. V. Mosby Co.) 2nd ed., pp. 283-369; 1977.

Immune deficiency states associated with a number of human malignancies are reviewed. Hodgkin's disease is associated with inability to express delayed hypersensitivity, inability to make circulating antibody, abnormally prolonged allograft rejection reactions, poor primary humoral antibody response, and intact secondary humoral antibody response. The immune deficiency state associated with acute leukemia is attributed to abnormal lymphocyte function and a reduction in total normal lymphoid tissue. Antibody production and delayed hypersensitivity reactions are impaired. Two populations of small lymphocytes are present in the peripheral blood of patients with chronic lymphocytic leukemia (CLL). One is composed of normal T and B lymphocytes, but the other is composed of leukemic cells that are incapable of normal immune responses. It is the gradual accumulation of this second population that leads to defective adaptive immunity in CLL. Patients with multiple myeloma have impaired ability to form humoral antibody; cellular immunity, however, is relatively intact. Several studies of immune function in patients with nonlymphoid solid tumors show a pattern of immunologic impairment similar to that seen in Hodgkin's disease. Immune deficiency states in human malignancy appear to result from intrinsic lymphocyte dysfunction or from humoral serum factors that interfere with what might otherwise be normal lymphocyte function. (922 refs.)

**77-3033 Immunology of Tumors in Experimental Animals.** (Eng.) Sinkovics, J. G. In: *The Immunology of Malignant Disease*. (St. Louis: C. V. Mosby Co.) 2nd ed., pp. 93-282; 1977.

Experimental investigations that have led to the practical application of tumor immunology in man are reviewed. Topics considered include antigens of virally and chemically induced tumors, embryonal tumor antigens, and immune reactions to tumors. Three major groups of mammalian oncornaviruses have been postulated: (1) the primate viruses, (2) the endogenous feline RD-114 virus, and (3) the feline, hamster, rat, and mouse leukemia-sarcoma viruses. Oncornaviruses appear to contain oncogenes in addition to virogenes; the presence of oncogenes has not been clearly demonstrated in oncogenic DNA viruses. Evidence favors the existence of immune surveillance; privileged sites deprived of lymph circulation have been shown to be increasingly susceptible to chemical carcinogenesis. Tumor cell products are poorly defined substances that either increase cell growth and migration or suppress the host's immune faculties. Cell fusion often, but not always, leads to loss of malignancy. Thus, neoplastic cells appear to have lost a "complementing component" that can be restored by hybridization with a nonmalignant partner. Some experiments imply that fusion between neoplastic and nonneoplastic cells may occur in vivo and that this mechanism may have a role in antitumor defense reactions. Probably the most important mechanism by which tumors outgrow the host's immune reactions is excessive shedding of cell membrane antigens. (1884 refs.)

**77-3034 Cell-mediated Immune Reactions to Human Tumors.** (Eng.) Perlmann, P. (Dept. Immunology, Univ. Stockholm, S10691, Stockholm, Sweden) Troye, M.; Pape, G. R. *Cancer (Suppl)* 40(1): 448-457; 1977.

Cell-mediated immune reactions to tumors, as demonstrated by skin testing or different in vitro procedures, occur in a variety of human neoplastic diseases. Studies of the cytotoxic effects of patients' blood lymphocytes constitute the most widely used in vitro test system. Although these tests provide some evidence for the occurrence of disease-related lymphocyte reactions in cancer patients, the results are controversial. The problems involved are exemplified by the results of an analysis of lymphocyte cytotoxicity in human urinary bladder carcinoma. Here, lymphocyte cytotoxicity to tumor cells frequently reflects the responses against a variety of antigens expressed to different degrees on tumor cells of different types. Although some of these antigens probably are tumor-associated, others are not, and they may give rise to immune responses in healthy individuals as well. Moreover, the response pattern varies in different individuals with the course of disease and after therapy. For this reason, disease-related reactions may well be detected in selected groups of patients but not readily in individual cases. This restricts the usefulness of the assay as a prognostic or diagnostic tool for monitoring cancer patients. That cell-mediated cytotoxicity



reflects activities of both antibody-dependent and antibody-independent effector cells further complicates the problem. Thus, cytolysis in vitro of allogeneic tumor cells is frequently mediated by a patient's lymphocytes acting in conjunction with humoral antibodies. On the other hand, as in certain experimental tumor systems, specific antibody-independent T cells may be the effector cells when autochthonous tumor cells are the target cells. In these instances other, more strictly tumor-specific antigens may be involved in the reactions than in antibody-dependent cellular cytotoxicity. Antibody-independent effector cells that kill tumor cells in a relatively nonselective manner may also be involved. Since these different mechanisms differ in importance for defense against tumor growth, further elucidation of the nature of effector cells and of the specificity of the cytotoxic reactions during different phases of disease appears indicated. This is particularly important in the manipulation of the cancer patient's immune response by immunotherapy. (71 refs.)

- 77-3035 **Innate Resistance and Specific Immunity in Host Control of Cancer.** (Eng.) Alexander, P. (Div. Tumor Immunology, Chester Beatty Res. Inst., Sutton, Surrey, England) *J Med* 8(3/4): 279-285; 1977.

The role of immune mechanisms in neoplasia is reviewed. The role of macrophages in innate resistance and the involvement of T lymphocytes are among the aspects covered. Innate resistance to sporadically occurring malignant cells may be exercised by mononuclear phagocytes. Although there is no support for the hypothesis that specific immune processes involving T lymphocytes determine the incidence of cancer, they influence the biological behavior and natural history of some tumors once they become clinically evident. (8 refs.)

- 77-3036 **Spontaneous Regression of Cancer: Summary and Profile for the Future.** (Eng.) Nossal, G. J. (Walter and Eliza Hall Inst. Medical Res., Melbourne, Victoria 3050, Australia) *Natl Cancer Inst Monogr* 44: 145-148; 1976.

The proceedings of the Conference on Spontaneous Regression of Cancer, held at The Johns Hopkins Medical Institutions, Baltimore, MD, on May 9 and 10, 1974, are summarized. Spontaneous regression of advanced cancer is extremely rare, occurring in < 1/1,000 cases. Furthermore, 60% of all regressions occur in only four cases: renal cell carcinoma, neuroblastoma, melanocarcinoma and choriocarcinoma. The following points relating to spontaneous regression are discussed: clinical documentation; surgery, radiotherapy, and psychological factors; established facts in immune responses to tumors; immunological events during tumor progression; and the future of tumor immunology. (1 ref.)

- 77-3037 **Burkitt's Lymphoma.** (Ger.) Rathke, W. (Facharzt für Augenkrankheiten, Geisenheimer Strasse 26, 6220 Rudesheim/Rhine, W. Germany) *Fortschr Med* 95(10): 608-614; 1977.

The epidemiology, clinical features, and treatment of Burkitt's lymphoma are discussed. Although Epstein-Barr virus has been incriminated in the pathogenesis of Burkitt's lymphoma, there is no experimental evidence that the virus is oncogenic in humans. (16 refs.)

- 77-3038 **Burkitt's Tumour: Lessons from Mice, Monkeys, and Man (Letter to Editor).** (Eng.) Mann, R. B. (Hematopathology Section, Lab. Pathology, NCI, NIH, Bethesda, MD 20014) Berard, C. W. *Lancet* 2(8028): 84; 1977.

Experimental results in mice suggest that the germinal centers of the mesenteric lymph node may represent the site of origin of Burkitt's tumor, with secondary spread to the intestine. Bilateral mammary involvement may appear in mice injected with the tumor during pregnancy and lactation. (7 refs.)

- 77-3039 **The Malignant Lymphomas.** (Eng.) Bloomfield, C. D.; Kennedy, B. J. In: *Management of the Patient with Cancer*. Nealon, T. F., ed. (Philadelphia: W.B. Saunders Co.) 2nd ed., pp. 917-963; 1976.

Current concepts of pathology and clinical staging of Hodgkin's disease and non-Hodgkin's malignant lymphoma are reviewed, and the management of patients with these diseases is presented. Etiologic roles have been suggested for viral, genetic, and immunologic factors, but how these factors interact remains unknown. There is general agreement on the histologic classification of Hodgkin's disease into four types: lymphocyte predominance, nodular sclerosis, mixed cellularity, and lymphocyte depletion. In most series, survival is best for lymphocyte predominance and poorest for lymphocyte depletion; survival for nodular sclerosis is usually much better than that for mixed cellularity. The three classifications for non-Hodgkin's lymphomas each identify four cell types: well-differentiated lymphocytes, poorly differentiated lymphocytes, histiocytes, and stem cells. The most common presenting manifestation is pain-free lymphadenopathy. Of the many systemic symptoms, three signify a poorer prognosis when present at diagnosis: unexplained fever of > 38 C, unexplained wt loss of > 10% of body wt in the previous 6 mo, and night sweats. Primary extranodal lymphomas are relatively unusual, but involvement of sites other than the lymph nodes is common. Clinical signs and symptoms are presented for the major extranodal areas, which include the

head and neck, intrathoracic region, gastrointestinal tract, liver, spleen, urinary and genital tracts, nervous system, bone, skin, and soft tissue. Most alterations in the peripheral blood of patients with malignant lymphomas are nonspecific, with anemia being the most frequent complaint. Patients with malignant lymphoma also have a variety of immunologic abnormalities. Progress in treatment has paralleled improvement in techniques for identifying the extent of disease. Treatment is by radio- or chemotherapy. (153 refs.)

- 77-3040 Reasons for Familial Aggregation in Hodgkin's Disease (Letter to Editor).** (Eng.) Grufferman, S. (Duke Univ. Medical Center, Durham, NC 27706) Cole, P.; Smith, P.; Lukes, R. J. *N Engl J Med* 296(16): 940-941; 1977.

A review of the literature and a small study indicated that the observed familial aggregation of Hodgkin's disease does not have a genetic basis. The study was too small to evaluate the incidence among spouses of affected persons. (no refs.)

- 77-3041 Certain Aspects of the Use of New Methods of Microscopy in Oncology.** (Eng.) Kraievski, N. A. (Dept. Pathological Anatomy Human Tumours, Oncological Res. Centre, Acad. Medical Sciences USSR, Moscow 115478 USSR) Raikhlin, N. T. *Folia Histochem Cytochem (Krakow)* 14(4): 211-216; 1976.

Light microscopy has shown that tumor cells in the process of development acquire simpler structures and undergo dedifferentiation. However, histochemical, immunological, and electron microscopic studies of tumors of the thyroid, liver, and urinary bladder, as well as melanomas, rhabdomyosarcomas, fibrosarcomas, and synoviomas, reveal that human malignant tumors retain sufficient characteristics to allow identity of the tumor origin. As an example, the thyroid gland possesses three types of cells that can correspondingly develop into three types of cancer with typical histochemical and ultrastructural properties of follicular cells (A cells), Ashkenazy cells (B cells) and parafollicular cells (C cells). Cancerous A cells are rich in ergastoplasm, cancerous B cells contain large numbers of mitochondria, and cancerous C cells are clear, with numerous vesicles and secretory granules. The wider use of new techniques, such as electron microscopy, in tumor diagnosis demands the greater attention of pathologists. (19 refs.)

- 77-3042 The Nervous System.** (Eng.) Morley, T. P. In: *Management of the Patient with Cancer.* Nealon,

T. F., ed. (Philadelphia: W. B. Saunders Co.) 2nd ed., pp. 759-802; 1976.

The pathology, diagnosis, and treatment of tumors of the nervous system are reviewed. The group of tumors known as gliomas range from the rapidly growing glioblastoma multiforme to the well-differentiated, slowly growing fibrillary astrocytoma, and they include the oligodendroglioma and ependymoma. These tumors are intrinsic to nervous tissue, invasive, and lack encapsulation, but they do not metastasize. Although rare, cerebellar hemangioblastoma is important because it is often curable by surgical excision. In metastatic tumors, the most common primary site is the lung, followed by the breast, kidney, gastrointestinal tract, and thyroid. Malignant melanoma frequently metastasizes to the brain. Radiographs of the skull and chest, computerized transaxial tomography, electroencephalography, echoencephalography, radiologic contrast studies (angiography, pneumography, and positive-contrast ventriculography), and gamma encephalography may aid in arriving at a correct diagnosis. The most common of the intrinsic tumors of the spinal cord is ependymoma, which usually appears in the cervical cord. Astrocytoma is an uncommon tumor, but it is important because of its innocence. In spinal meningiomas, the incidence of malignant features is very low and they do not recur after total gross removal. Most multiple spinal schwannomas occur as a part of von Recklinghausen's disease. Intramedullary deposits of metastatic carcinoma are very rare. Diagnosis may be assisted by plain radiography, myelography, and, in certain circumstances, lumbar puncture. Tumors of peripheral nerves are also briefly discussed. (30 refs.)

- 77-3043 Colonic Polyps: Antecedent- or Associated-Lesions of Large Bowel Cancer.** (Eng.) Rawson, R. W. (Dept. Scientific Operations, Natl. Large Bowel Cancer Project, Univ. of Texas System Cancer Center, Houston, TX 77030) *Semin Oncol* 3(4): 361-367; 1976.

This study discusses whether large bowel cancers arise in adenomatous polyps or in villous adenomas. Some pathologists even favor the thesis that colorectal cancers evolve de novo from normal colonic mucosa. Familial polyposis coli and Gardner's syndrome are premalignant and, if untreated, lead to the development of one or more adenocarcinomas in the large bowel. They should be treated surgically by subtotal colectomy with ileoproctostomy and fulguration of polyps remaining in the rectal segment. Many pathologists, gastroenterologists, and oncologists agree that the villous (papillary) adenomas of the large bowel are at risk for the development of large bowel cancer. The controversy over the role of adenomatous polyps (tubular adenomas) in the genesis of large bowel cancer cannot yet be decided. Polyps larger than 1.0 cm should be removed through the proctosigmoidoscope or the colonoscope. (33 refs.)



- 77-3044 **Histogenesis of Salivary Gland Neoplasms.** (Eng.) Regezi, J. A. (Dept. Oral Pathology, Univ. Michigan Sch. Dentistry, Ann Arbor, MI 48109) Bat-sakis, J. G. *Otolaryngol Clin North Am* 10(2): 297-307; 1977.

Evidence from light and electron microscopy studies supporting a hypothetical bicellular theory of origin of salivary gland neoplasms from undifferentiated excretory duct reserve cells or intercalated duct reserve cells is reviewed. The latter cells may be the progenitor of both ductal and myoepithelial cells. In mixed tumors, neoplastic myoepithelial cells appear to undergo mesenchymal metaplasia. These myoepithelial cells may also be responsible for the wide microscopic variety of salivary gland tumors. The ultimate cell of origin for oncocytic tumors, adenoid cystic carcinoma, acinic cell carcinoma, and adenocarcinoma also appears to be the intercalated duct cell. Direct neoplastic transformation of the excretory duct reserve cell, which normally differentiates into squamous, columnar, and mucous cells of the excretory duct, could result in squamous cell or mucoepidermoid carcinoma. (11 refs.)

- 77-3045 **Teratomas and Yolk-Sac Tumours.** (Eng.) Brown, N. J. (Royal Hosp. for Sick Children, Bristol, England) *J Clin Pathol* 29(11): 1021-1025; 1976.

The origin of teratomas and yolk-sac tumors is discussed based on a working hypothesis that these neoplasms arise from germ cells. This hypothesis suggests that during embryonic migration some germ cells may be left behind or may stray too far and come to rest at various sites along the dorsal wall of the embryo near the midline. If these cells fail to degenerate and remain viable, they may give rise to tumors in the retroperitoneum, sacral region, mediastinum, and pineal region. The histologic and diagnostic features of testicular, ovarian and sacrococcygeal teratomas, other extragonadal teratomas, yolk-sac tumors of the testis and the ovary, and extragonadal yolk-sac tumors are discussed. (7 refs.)

- 77-3046 **Biology of Breast Preneoplasia.** (Eng.) Cardiff, R. D. (Dept. Pathology, Sch. Medicine, Univ. California, Davis, CA 95616) Wellings, S. R.; Faulkin, L. J. *Cancer (Suppl)* 39(6): 2734-2746; 1977.

The literature dealing with mammary cancer and the presence of identifiable preneoplastic lesions in the mammary gland of several species is reviewed. A discrete morphologically identifiable lesion with a high malignant potential is considered preneoplastic. Lobuloalveolar lesions found in the human breast fit most of the criteria for preneoplasia. The lesions, hyperplastic atypical lobules, are multicentric, have a high statistical association with breast cancer, and demon-

strate a morphological progression through dysplasia to frank carcinoma. Other breast lesions have a high statistical association but no morphological progression can be seen and their neoplastic potential remains unknown. Statistical correlation with mammary cancer and aberrant morphology has been accepted as presumptive evidence of preneoplasia in some animals. Definitive proof of the neoplastic potential has been obtained in rodent models by transplantation of the suspected lesions and their subsequent malignant transformation. Although a strong morphological and statistical analogy exists between the human and animal models, the causes and biological potential of the human lesions await experimental proof. (51 refs.)

- 77-3047 **The Role of Hormones in the Etiology of Human Breast Cancer.** (Eng.) Kirschner, M. A. (Newark Beth Israel Medical Center, 201 Lyons Ave., Newark, NJ 07112) *Cancer (Suppl)* 39(6): 2716-2726; 1977.

Epidemiologic and clinical observations on the role of endocrine factors in breast cancer are reviewed. Women at increased risk for breast cancer excrete less estriol ( $E_3$ ) vs estrone and estradiol. Conversely, women at low risk for breast cancer had higher urinary estriol ratios. These data led to the hypothesis that  $E_3$  is a "protective" estrogen. Recent studies of estriol production rates, its origin, and circulating levels, however, have shown no differences in these crucial parameters in women with high  $E_3$  ratios vs low  $E_3$  ratios or in women with previous breast cancer. These data imply that high vs low urinary  $E_3$  ratios simply reflect different pathways of estrogen metabolism and not differences in estrogen production. In women with established breast cancer, earlier studies of urinary estrogens as well as recent measurements of estrogen blood production rates showed no significant abnormalities. Further, the contribution of androstenedione; estrone's principal prehormone, is normal in women with breast cancer. Earlier studies in women with breast cancer correlated decreased urinary 17-ketosteroids with poor responses to endocrine ablative procedures. These studies also found low urinary ethiocholanolane in women destined to develop breast cancer. Thus, decreased androgen excretion seems to be associated with poor response to endocrine therapy and increased risk for breast cancer. Similarly, dehydroepiandrosterone and its metabolites were decreased in women with breast cancer. Androstenedione production, however, was normal in women with postmenopausal breast cancer. The significance of androgen alterations in women with breast cancer (or at high risk) is still unclear. No abnormalities in progesterone production have been reported in women with breast cancer. The Sherman-Korenman hypothesis that women with inadequate corpus luteum formation are at increased risk for breast cancer still requires confirmation. Most reports to date indicate no prolactin abnormalities in women with breast cancer. Data showing high plasma prolactin and estradiol in daughters of women with breast cancer (high risk) need confirmation. In spite of several intriguing

associations of thyroid function with steroid and peptide hormone metabolism, no consistent evidence has emerged implicating abnormal thyroid function in women with breast cancer. In spite of fragmentary data linking breast cancer development with possible alteration of endocrine function, no clear-cut or comprehensible patterns are yet evident. (130 refs.)

**77-3048 Genetic Factors in the Etiology of Breast Cancer.** (Eng.) Petrakis, N. L. (George Williams Hooper Foundation, Univ. California, San Francisco, CA 94143) *Cancer (Suppl)* 39(6): 2709-2715; 1977.

Human breast cancer appears to be a heterogeneous disease whose causes are still unknown. A variety of interrelated genetic, environmental, sociobiologic, and physiological factors appear to be associated with increased risk of breast cancer, but no single factor or combination of variables presently known are sufficient to explain its etiology. Evidence for a genetic role in susceptibility to breast cancer is based on findings from several lines of investigation, including: (1) family history of breast cancer, especially bilaterality; (2) marked difference in rates among certain racial groups; (3) lack of major change in incidence of breast cancer over many years despite dramatic changes in other cancers; (4) concordance of breast cancer in monozygotic twins; and (5) concordance of laterality of breast cancer in closely related patients. Recognition of familiarity and bilaterality has important clinical value in identifying high-risk women for special screening and diagnostic studies. It is not known if a single gene or several genetic mutations are responsible for a specific predisposition to breast cancer. Possible genetic traits that control or influence physiological functions of the breast may be inherited by certain women. These genetic factors that increase or decrease the probability of neoplastic transformation may operate through their control over viral, hormonal or other stimuli and through their regulation of the response of the alveolar-ductal epithelium of the breast to these stimuli and to secreted extrinsic carcinogens. A working hypothesis has been developed that interrelates genetic-environmental interactions in breast cancer etiology and pathogenesis. In this model, the turnover rate of breast secretions is the primary determinant of the extent and duration of exposure of the breast epithelium to extrinsic and endogenous carcinogens. The model emphasizes the interaction of genetic, physiological, endocrine, and environmental factors in the epidemiology of breast cancer. (45 refs.)

**77-3049 Genetic Etiology of Cancer.** (Eng.) Strong, L. C. (Section Medical Genetics, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) *Cancer (Suppl)* 40(1): 438-444; 1977.

Cancer may be a genetic disease at the cellular level. Studies of chemical, radiation, and viral carcinogenesis, as well as of cytogenetics and hereditary cancers, suggest that irreversible changes in the cellular hereditary material occur in the development of a cancer cell. Hereditary or environmental factors may influence the probability of such changes. A small fraction of human cancers may be attributed to rare hereditary disorders of chromosomal abnormality, mutagenesis, cell growth regulation or differentiation, or immune deficiency. Yet studies of familial cancer suggest that in the absence of such demonstrable abnormalities, a predisposition to cancer may follow an autosomal dominant hereditary pattern, as a result of which tumors that occur at relatively early ages and that may be multiple are generated. The occurrence of hereditary and nonhereditary forms of the same histologic cancer has been attributed to the occurrence of similar genetic changes in germinal or somatic cells, respectively. The percentage of tumors arising from germinal mutations varies for each tumor type, but it may account for nearly 40% of certain childhood cancers and a lesser percentage of adult cancers. Study of the interaction of hereditary and acquired risk factors suggests that more than a single genetic change may be necessary for cancer to develop. If a series of mutations are necessary, then those individuals with the inherited mutation present in all cells may be uniquely susceptible to carcinogens. Individuals genetically predisposed to cancer may be identified through family studies, recognition of associated anomalies, or follow-up of patients with a previous cancer. Appropriate clinical examinations may lead to early cancer detection, and research studies may reveal genetic markers or early cellular changes predisposing to cancer. (66 refs.)

**77-3050 Genetic Risks for Human Breast Cancer.** (Eng.) Anderson, D. E. (Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) *Cancer Detect Prevent* 1(2): 283-291; 1976.

The relationship of family history and breast cancer incidence is discussed. The risk of premenopausal bilateral breast cancer is 8.8-fold greater among first-degree relatives of breast cancer victims than among control subjects. Conversely, the risk of postmenopausal unilateral disease among these relatives is only 1.2-fold greater than that for controls. Analyses of breast cancer data for the mothers and sisters of victims suggest a hereditary type of breast cancer in which predisposition is vertically transmitted from mother to daughter, so that the daughters have a 30%-35% probability of breast cancer development during their lifetime. The concept of a hereditary form of the disease is supported by the following observations: (1) women at risk through their pedigree develop the disease at earlier ages than usual; (2) the disease in high-risk pedigrees is frequently bilateral (12%-15% versus 3% in unselected patients); (3) the disease develops at similar ages in sisters; (4) the lifetime probability of 30%-35% is close to that expected (50%) with a dominant inheritance pattern. There is a close similarity in disease frequency for



both maternal and paternal lines of descent: this argues against the possibility that the hereditary form of the disease is caused by a milk-transmitted virus. Indeed, the hereditary form of the disease could be associated with ovarian estrogens, since the risks are highest when the ovaries are most active and then decline when the ovaries become less active. (9 refs.)

- 77-3051 **Epidemiology of Cancer of the Penis.** (Eng.) Persky, L. (Univ. Hosps., Dept. Urology, 2065 Adelbert Rd., Cleveland, OH 44106) *Recent Results Cancer Res* 60: 97-109; 1977.

Worldwide incidences of penile cancer are reviewed and interpreted with respect to several epidemiologic factors. The lowest rates appear in groups practicing infant circumcision and in areas where standards of sexual hygiene are high. Phimosis is the most common precancerous condition; high rates of congenital phimosis have been associated with high rates of penile cancer. The cancer-inducing potential of smegma (the production of which begins during the first days of life) has been suggested to account for the reduced effectiveness of circumcision performed after the neonatal period in preventing penile cancer. In some studies venereal disease has been implicated in the etiology of the disease, but others have found that it does not play an important role. Hot, humid climates seem to favor development of penile cancer. Paget's disease may be a predisposing factor in some cases. Subnormal immunologic responses have been reported in about 50% of patients with penile cancer. It has been suggested that viral interaction might cause verrucous carcinoma of the penis. (79 refs.)

- 77-3052 **Epidemiology of Prostate Carcinoma.** (Eng.) Tulinius, H. (Icelandic Cancer Society, Post Office Box 523, Reykjavik, Iceland) *Recent Results Cancer Res* 60: 3-13; 1977.

Mortality and incidence rates for carcinoma of the prostate are given, along with some suggestions as to its etiology. Prostate carcinoma is numerically one of the most important malignant causes of death in man in northwestern Europe and North America. Chinese and Japanese populations have very low rates. The significant frequency starts at age 60, after which the incidence curve rises steeply to the highest age groups. The incidence of prostatic carcinoma is now rising in all age groups. Almost nothing is known about the risk factors important for this disease. The strong correlation with age suggests either an unusually long latent period or that the risk factors start to operate relatively late in life. Diet, air pollution, hormones, and cadmium have been suggested

as etiologic agents. Sexual promiscuity has also been associated with an increased risk of prostatic carcinoma. If this association is confirmed, two explanations could be offered: the first would implicate an infectious agent or therapy for venereal diseases; the second relates to alteration of the chemical composition of semen, which would implicate cadmium. (31 refs.)

- 77-3053 **Diet and Cancer of Endocrine Target Organs.** (Eng.) Cole, P. (Dept. Epidemiology, Harvard Sch. Public Health, 677 Huntington Ave., Boston, MA 02115) Cramer, D. *Cancer (Suppl)* 40(1): 434-437; 1977.

The role of diet in the etiology of breast and endometrial cancer is considered. Geographic variation in rates, effects of migration, consequences of exogenous hormone use, and other epidemiologic factors are reviewed. Endometrial cancer is related to diet, probably through simple caloric excess. The excess is reflected in obesity and the consequent overproduction of estrone, a natural but carcinogenic human estrogen. The same mechanism may operate in breast cancer, but it does not explain a large proportion of the disease. Some specific dietary factor, for example, saturated fat, may be causally related to breast cancer. Such a relationship would probably be mediated through an endocrine mechanism. (26 refs.)

- 77-3054 **Colon Cancer: Epidemiology.** (Eng.) Walker, A. R. (South African Medical Res. Council, Human Biochemistry Res. Unit, South African Inst. Medical Res., P.O. Box 1038, Johannesburg, 2000, South Africa) Burkitt, D. P. *Semin Oncol* 3(4): 341-350; 1976.

The epidemiology of cancer of the colon as it prevails in western urban populations, primitive and developing populations, and immigrant populations is discussed. The magnitude of the influence of ethnic and individual susceptibility is unknown. The disease occurs principally in late middle age and old age and is more prevalent among those with high incomes who consume a westernized diet. In western populations, colonic cancer mortality rates are slightly greater in men. For Chinese immigrants in the US, the mortality from cancer of the colon is much higher in men than in women. Among Rhodesian and Ugandan blacks, the figure is higher in women than in men. Evidence indicates that the main cause of colon cancer is a change in diet. Data suggest that noxious metabolites are produced in the bowel from interactions between digesta and intestinal bacteria. Changes in the westernized diet include increases in calories, sugar, protein, and fat and a reduction in the intake of crude fiber and bulk-forming material. In black African populations with low incidences of colon cancer, bowel behavior differed significantly from western populations. Defecation was more frequent, the

amount of feces voided was greater, transit time was shorter, and the feces were soft and passed with ease. In nonprone populations, the feces contain lower levels of nitrogen, fat, cholesterol, bile acids, and sterols. A lower degree of degradation of these compounds to potentially carcinogenic metabolites is also observed. High risk populations were discovered to contain more anaerobic bacteria in the feces than the non-prone populations. A hypothesis of colonic cancer causation and preventive measures is discussed. (60 refs.)

77-3055 **Epidemiology of Cancer: Current Perspectives.** (Eng.) Doll, R. (Oxford Univ., Oxford OX2 0PS, England) *Am J Epidemiol* 104(4): 396-404; 1976.

Epidemiological studies of cancer have made a valuable contribution to the current state of knowledge for several reasons. First, they have helped define the environmental conditions responsible for the production of the disease; second, they have provided indications for the possibility of prevention; and third, they have monitored the effects of intervention to prevent the disease. Such studies may also aid in understanding the mechanisms of carcinogenesis. Past contributions in the history of epidemiology in cancer research, covering the importance of age, environment, diet and biological agents, are reviewed. Epidemiology can be of value in further detailed mapping of cancer incidence by the establishment of cancer registries for linking personal, occupational, and medical histories and death rates. This latter idea could well be opposed by the layman who is concerned with the extensive use of computerized files, but such files are necessary if the public is to have the protection it wants against cancer from drugs and industrial products. (26 refs.)

77-3056 **Discussion of: "Epidemiology of Cancer: Current Perspectives".** (Eng.) Levin, M. L. (Dept. Epidemiology, Sch. Hygiene and Public Health, The Johns Hopkins Univ., Baltimore, MD 21205) *Am J Epidemiol* 104(4): 405-407; 1976.

Perspectives on the epidemiology of cancer are reviewed in relation to a recently published paper. In spite of increased knowledge of environmental causes of cancer, there has been no reduction in the occurrence of cancers, except for gastric cancer. A 40-50% reduction in the incidence of cancer would be more realistic than previous estimations of an 80-90% reduction. The reduction should occur chiefly among men, due to a decrease in smoking. Occupational cancers are difficult to eliminate because of economic considerations. It should be possible to avoid the great majority of iatrogenic cancers if the use of potentially damaging and carcinogenic drugs is restricted. Epidemiology ought to be substantially enlarged, including monitoring and control of the effectiveness and maintenance of preventive measures, in addition to early screening. (6 refs.)

77-3057 **Importance of Environmental Factors in Cancer.** (Eng.) Higginson, J. In: *Environmental Pollution and Carcinogenic Risks*. (Lyon: International Agency for Research on Cancer): IARC Scientific Publications No. 13, INSERM Symposia Series Vol. 52, pp. 15-23; 1976.

The role of the epidemiologic method in the identification of carcinogenic hazards in the environment is reviewed. Apart from the cancers that are known to be caused by occupation (eg, lung cancer among asbestos workers) or specific drugs, the evidence of the importance of the environmental background in human cancer is based mostly on geographic variations in incidence and on changes in cancer incidence among people who migrated. The most important cancers of known etiology are those dependent on a cultural habit, eg, smoking. Apart from skin pigmentation and individual susceptibility, congenital and hereditary factors are of little significance. It was found to be more useful to compare the risk of developing cancer over a life-span rather than just in age-specific groups. The causes of all cancers have not yet been discovered, but the evidence points to a large number of them being dependent on environmental factors. (24 refs.)

77-3058 **A Review of Possible Effects of Soil, Water and Meteorological Factors on Cancer.** (Eng.) Tromp, S. W. (International Society of Biometeorology, Jan Steenlaan 3, Oegstgeest, Leiden, Netherlands) *Med Biol Environ* 4(1): 66-74; 1976.

Epidemiological data concerning the effects of soil, water supply, and weather on cancer incidence are summarized. Soil factors include the location of living quarters, soil types, trace elements, and geophysical fields (in particular, low-resistivity zones in soil). Two studies relevant to the effects of water supply are discussed: (1) a study in Greater London in 1947, demonstrating different incidences of all cancers and stomach cancer in regions supplied by wells or by river; (2) a Dutch study in 1953, demonstrating a higher overall cancer mortality in municipalities without a water system compared to those with a system (mortalities of 606 vs 543/100,000). The influence of the chemical composition of drinking water is mentioned. Weather factors include thermal and seasonal effects, the effects of high altitudes, pollution, the indirect effect of meteorological factors on oncogenic viruses, and physiological changes in the body due to meteorological stresses. (96 refs.)

77-3059 **Ethnic Background and Cancer in Israel.** (Eng.) Modan, B. (Dept. Clinical Epidemiology, Chaim Sheba Medical Center, Tel Hashomer and Tel Aviv Univ. Medical Sch., Israel) *Med Biol Environ* 4(1): 3; 1976.

European-born Israelis have a greater risk of developing can-



cer than do immigrants from the Middle East or North Africa. Because the increased risk involves primarily cancers of the gastrointestinal and reproductive systems, nutritional factors must be considered as etiologic agents. (no refs.)

- 77-3060 **Diseases in Urban and Rural Black Populations.** (Eng.) Seftel, H. C. (Dept. Medicine, Johannesburg Non-European Hosp. and Univ. Witwatersrand, Johannesburg, South Africa) *S Afr Med J* 51(5): 121-123; 1977.

Esophageal cancer and hepatic cancer are found more often in rural blacks in South Africa than in urban blacks, although esophageal cancer is the most common cancer among black males in Johannesburg. Cancer of the uterine cervix is equally prevalent in both localities. Other diseases are also discussed. (no refs.)

- 77-3061 **Summation: Molecular Mechanisms of Gene Regulation--Session 3.** (Eng.) Weinhouse, S. (Fels Research Inst., Temple Univ. Sch. Medicine, Philadelphia, PA 19140) *Cancer Res* 36(11): 4330-4331; 1976.

The molecular mechanisms of gene regulation and in particular the appearance of fetal isozymes in cancer are discussed based on previous work with Morris hepatomas and the hexokinases, aldolases, pyruvate kinases, adenylate kinases and glycogen phosphorylases. Since the activation of fetal isozymes is accompanied by the inactivation of adult genes, the switch in gene expression may be the molecular basis for the loss of control of cell proliferation in cancer. Whatever functional means are used for the identification of proteins, tumors are found to express proteins that have been suppressed during normal tissue differentiation. Many of the properties of the neoplastically transformed cells may well be due to a massive alteration of protein composition. (9 refs.)

- 77-3062 **Expression of an Oncodevelopmental Gene Product ( $\alpha$ -Fetoprotein) During Fetal Development and Adult Oncogenesis.** (Eng.) Sell, S. (Dept. Pathology, Univ. California San Diego Medical Sch., La Jolla, CA 92093, USA) Becker, F. F.; Leffert, L. H.; Watabe, H. *Cancer Res* 36(11): 4239-4249; 1976.

The production of  $\alpha$ -fetoprotein (AFP) in normal development, in regeneration of adult liver, by fetal rat hepatocytes in vitro, by animals bearing transplantable hepatomas, and by animals fed chemical carcinogens is reviewed. AFP is a serum protein made normally during fetal and neonatal stages by liver and yolk sac cells. In neonatal rats, AFP pro-

duction is terminated at about 4 wk of age; this corresponds with the cessation of liver cell proliferation. Elevated concentrations of serum AFP are observed following events leading to restitutive liver regeneration, such as hepatectomy. In vitro studies utilizing combined autoradiography for DNA-synthesizing cells and immunofluorescence for AFP-containing cells demonstrate that replicating hepatocytes produce AFP. Serum concentrations of AFP may be used as an accurate index of the growth of an AFP-producing tumor. If the tumor is removed, AFP production can be used as a marker of metastatic growth. Exposure to chemical hepatocarcinogens leads to a sequence of morphologic and biochemical changes culminating in the development of malignant hepatocellular carcinomas. This exposure results in the appearance of elevated serum AFP concentrations as early as within 1 wk of feeding. AFP is not a general immunosuppressive, although some in vitro immune responses are affected by AFP. The production of AFP is associated with normal hepatocyte division in both fetal and adult rats and with exposure to chemical carcinogens in the presence or absence of hepatocyte proliferation. (83 refs.)

- 77-3063 **Interrelationships and Functions of the Pyruvate Kinase Isozymes and Their Variant Forms: A Review.** (Eng.) Ibsen, K. H. (Dept. Biological Chemistry, California Coll. Medicine, Univ. California, Irvine, CA 92717) *Cancer Res* 37(2): 341-353; 1977.

The relationships among the properties of pyruvate kinase isozymes are reviewed, with emphasis on their potential role in carcinogenesis. Particular consideration is given to: evaluation of the concept that the three major nonreadily interconvertible forms (K, L, and M isozymes) are the products of distinct genes; the relationship of these forms to additional separable forms of pyruvate kinase; the types and possible functions of interconvertible forms of the major isozymes; and mechanisms affecting the genetic expression of the isozymes. The apparent derepression of the fetal isozyme in hepatomas and the influence of tumor extracts on pyruvate kinase expression in the liver of host animals are discussed. (197 refs.)

- 77-3064 **NGF May Hold the Key--But to What?** (Eng.) Arehart-Treichel, J. (No affiliation given) *Sci News* 11(21): 330-331; 1977.

Many tumor cells appear to secrete nerve growth factor (NGF), but its precise role in carcinogenesis is unknown. The presence of NGF in various tissues is discussed, along with the finding that melanoma cells appear to need NGF to survive. (1 ref.)

## CHEMICAL CARCINOGENESIS

- 77-3065 **Aflatoxicol M<sub>1</sub>, a New Metabolite of Aflatoxicol.** (Eng.) Salhab, A. S. (Dept. Pharmacology, George Washington Univ. Medical Sch., 2300 Eye St., NW, Washington, DC 20037) Abramson, F. P.; Geelhoed, G. W.; Edwards, G. S. *Xenobiotica* 7(7): 401-408; 1977.

A new metabolite of aflatoxicol, aflatoxicol M<sub>1</sub>, was isolated from the postmitochondrial and microsomal fractions of dog liver fortified with NADPH. This metabolic transformation is similar to the conversion of aflatoxin B<sub>1</sub> to aflatoxin M<sub>1</sub>. Aflatoxicol M<sub>1</sub> can also be produced from aflatoxin M<sub>1</sub> by a reductase present in rabbit liver cytosol, a reaction analogous to the reduction of aflatoxin B<sub>1</sub> to aflatoxicol. In addition, aflatoxicol M<sub>1</sub> can be oxidized to aflatoxin M<sub>1</sub> by a carbon monoxide-insensitive dehydrogenase activity associated with human liver microsomes, as in the production of aflatoxin B<sub>1</sub> from aflatoxicol by this fraction. The UV, fluorescence, and mass spectral characteristics of aflatoxicol M<sub>1</sub> are described. The possibility that this new aflatoxin metabolite is both toxic and carcinogenic is discussed. (14 refs.)

- 77-3066 **The Presence and Concentration of Aflatoxin M<sub>1</sub> in Milk Shipped to a Dairy Plant.** (Ger.)

Kiermeier, F. (Suddeutsche Versuchs- und Forschungsanstalt für Milchwirtschaft, Vottinger Strasse 45, D-8050 Freising-Weihenstephan, W. Germany) Weiss, G.; Behringer, G.; Miller, M.; Ranfft, K. *Z Lebensm Unters Forsch* 163(3): 171-174; 1977.

Thin-layer chromatography and mass spectrometry established the presence of aflatoxin M<sub>1</sub> in 79/419 milk samples. Of these 79 samples, 0.05-0.1 µg/liter was found in 33% and ≥ 0.1 µg/liter in 38% (av 0.12 µg/liter). The other aflatoxins were absent. A total of 43% of the feed samples taken from dairy farms with aflatoxin M<sub>1</sub>-positive milk were positive for aflatoxin B<sub>1</sub>. It was not possible to establish a quantitative correlation between aflatoxin M<sub>1</sub> concentration in milk and aflatoxin B<sub>1</sub> concentration in feed. (12 refs.)

- 77-3067 **The Presence and Concentration of Aflatoxin M<sub>1</sub> in Commercial Cheese Samples.** (Ger.)

Kiermeier, F. (Suddeutsche Versuchs- und Forschungsanstalt für Milchwirtschaft, Vottinger Strasse 45, 5050 Freising-Weihenstephan, W. Germany) Weiss, G.; Behringer, G.; Miller, M. *Lebensmittel Unters Forsch* 163(4): 268-271; 1977.

Thin-layer chromatography and mass spectrometry showed

that of 197 cheese samples (approx 20 different varieties), 136 were aflatoxin M<sub>1</sub> (AFM<sub>1</sub>)-positive. The max level was 0.23 µg/kg, and the av level was 0.09 µg/kg. Of these positive samples, 54% showed only traces of AFM<sub>1</sub>, 21% up to 0.1 µg/kg and 14% > 0.1 µg/kg. Soft cheese had less AFM<sub>1</sub> than the other varieties. Supplemental spring feeding of cows increased the AFM<sub>1</sub> concentration in the cheeses, particularly the soft types. (15 refs.)

- 77-3068 **Repair of DNA in Human Cells After Treatment with Activated Aflatoxin B<sub>1</sub>.** (Eng.) Sara-

sin, A. R. (Institut de Recherches Scientifiques sur le Cancer, Boite Postale 8, 94800 Villejuif, France) Smith, C. A.; Hanawalt, P. C. *Cancer Res* 37(6): 1786-1793; 1977.

Repair replication was studied in cultured human cells exposed to aflatoxin B<sub>1</sub> metabolically activated with microsomes from phenobarbital-pretreated Wistar rats. Repair replication was stimulated in WI38 diploid fibroblasts and in their simian virus 40 (SV40) transformants, VA13 cells, up to levels about 20% of that obtained after exposure to saturation doses of UV light. Repair replication was not stimulated by nonactivated aflatoxin B<sub>1</sub>. The time course of repair synthesis was similar to that seen after UV irradiation, and most of the synthesis was in "short patches" 30-50 nucleotides long. A line of SV40-transformed xeroderma pigmentosum cells (Group A), deficient in repair after exposure to UV light, was also deficient in repair replication after aflatoxin treatment. This indicates that the mechanisms involved in the repair of UV light- and aflatoxin-induced damage share at least one common step, probably the recognition of damage. Treatment with aflatoxin resulted in a 25%-45% inhibition of UV light-induced repair replication. Thus, the toxin may inhibit excision repair as well as produce lesions in the DNA that are substrates for the process. (41 refs.)

- 77-3069 **Biosynthesis of Aflatoxin B<sub>1</sub>. Conversion of Versicolorin A to Aflatoxin B<sub>1</sub> by *Aspergillus parasiticus*.** (Eng.) Lee, L. S. (Southern Region Res. Center,

Agricultural Res. Service, US Dept. Agriculture, New Orleans, LA 70179) Bennett, J. W.; Cucullu, A. F.; Ory, R. L. *J Agric Food Chem* 24(6): 1167-1170; 1976.

An investigation was made of the conversion of <sup>14</sup>C-versicolorin A (VA), a C-18 polyketide-derived hydroxyanthraquinone, to the mycotoxin and potent hepatocarcinogen aflatoxin B<sub>1</sub> (AB<sub>1</sub>) by resting cell cultures of wild-type *Asper-*



*gillus parasiticus*. The purpose of the investigation was to substantiate the suggestion that VA is a natural biosynthetic precursor to AB<sub>1</sub>. A high level of incorporation (46%) of VA into AB<sub>1</sub> was observed, providing evidence to support the suggestion. It also indicates that the radiolabeled VA was incorporated intact into AB<sub>1</sub> and not broken down into acetate units prior to incorporation. (22 refs.)

- 77-3070 **Biosynthesis of Aflatoxin. Conversion of Norsolorinic Acid and Other Hypothetical Intermediates into Aflatoxin B<sub>1</sub>.** (Eng.) Hsieh, D. P. (Dept. Environmental Toxicology, Univ. California, Davis, CA 95616) Lin, M. T.; Yao, R. C.; Singh, R. *J Agric Food Chem* 24(6): 1170-1174; 1976.

An investigation was made of the possible involvement of norsolorinic acid (NA) and derivatives of C18-anthraquinone (AQ) and anthrone (AT) as biosynthetic intermediates of aflatoxin B<sub>1</sub> (AB<sub>1</sub>). Ring-labeled <sup>14</sup>C-NA and four tritiated derivatives of AQ and AT were incubated with resting mycelium of the AB<sub>1</sub>-producing strain of *Aspergillus parasiticus*. Over 2% of the label from NA was accountable in repeatedly purified AB<sub>1</sub>, which possessed a specific activity of 0.19 relative to that of NA. Only trace amounts of label from the AQ and AT derivatives were seen in the purified AB<sub>1</sub>. It is concluded that the biosynthesis of AB<sub>1</sub> involves NA, but none of the derivatives of AQ and AT, as an intermediate. (28 refs.)

- 77-3071 **Induction of Mixed Function Oxidases on Oral Administration of Dieldrin.** (Eng.) Kohli, K. K. (Dept. Biochemistry, Vallabhbhai Patel Chest Inst., Univ. Delhi, Delhi-1 10007, India) Maggon, K. K.; Venkitasubramanian, T. A. *Chem Biol Interact* 17(3): 249-255; 1977.

The effect of dieldrin on the metabolism of aflatoxins B<sub>1</sub> and G<sub>1</sub> by male Wistar rats was investigated in liver microsomal preparations from treated animals. The metabolism of the aflatoxins was increased, indicating an increased mixed function hydroxylase activity. This could be due to an increase in protein synthesis. (37 refs.)

- 77-3072 **Mechanism of Aflatoxin B<sub>1</sub> Inhibition of Rat Liver Nuclear RNA Synthesis (Meeting Abstract).** (Eng.) Yu, F. (Thomas Jefferson Univ., Philadelphia, PA 19107) Fang, J.; Wisniewski, D. *Proc Am Assoc Cancer Res* 18: 88; 1977. (no refs.)

- 77-3073 **Influence of the Aryl Group on the Reaction of Glucuronides of N-Arylacethydroxamic Acids with Polynucleotides.** (Eng.) Irving, C. C. (Dept. Urology, Univ. Tennessee Center for Health Sciences, Memphis, TN 38104) *Cancer Res* 37(2): 524-528; 1977.

The reactions of the glucuronide (GU) conjugates of the carcinogens N-hydroxy-2-acetylaminofluorene (N-hydroxy-AAF), N-hydroxy-4-acetylaminostilbene (N-hydroxy-AAS), N-hydroxy-4-acetylaminobiphenyl (N-hydroxy-AABP), and N-hydroxy-2-acetylaminophenanthrene (N-hydroxy-AAP) with transfer RNA, ribosomal RNA, DNA polyadenylate, polyguanylate, polyuridylylate, polycytidylylate, poly(adenylate, guanylate), and poly(guanylate, uridylylate) were studied. The relative order of reactivity of these GU's with nucleic acids, measured by the covalent binding of the aryl group labeled with <sup>3</sup>H or <sup>14</sup>C, was GU of N-hydroxy-AAF > GU of N-hydroxy-AAS > GU of N-hydroxy-AABP > GU of N-hydroxy-AAP. The GU of N-hydroxy-AAP showed only marginal or negligible reactivity. The GU of N-hydroxy-AAF showed greater reactivity with polyguanylate than with polyadenylate, but the reverse was true for the GU of N-hydroxy-AAS. Both of these GU's had much lower extents of reaction with polyuridylylate and polycytidylylate. Except for the reaction of the GU of N-hydroxy-AABP with polyadenylate, there was no detectable reaction of this GU or the GU of N-hydroxy-AAP with the homopolynucleotides. Under comparable conditions, the GU conjugates of N-hydroxy-AAF, N-hydroxy-AAS, and N-hydroxy-AABP demonstrated greater reactivity with poly(adenylate, guanylate) and poly(guanylate, uridylylate) than with the homopolynucleotides. The synthesis of two new GU conjugates, those of N-hydroxy-AAS and N-hydroxy-AAP, is also described. (26 refs.)

- 77-3074 **Early Changes in Hepatic RNA Synthesis and Distribution in Rats Fed Acetylaminofluorene (Meeting Abstract).** (Eng.) Austin, G. E. (UCLA, Los Angeles, CA 90024) Moyer, F. H. *Proc Am Assoc Cancer Res* 18: 78; 1977. (no refs.)

- 77-3075 **Hormone Dependence of Urogenital Tumors Induced in Rabbits with 2-AAF (Meeting Abstract).** (Eng.) Coogan, P. S. (Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL) Miller, A. W.; Pellettiere, E. V.; Newton, J. J.; Hass, G. M. *Am J Pathol* 86(2): 26a-27a; 1977. (no refs.)

- 77-3076 **Species Difference in TA1538 Mutagenicity by Hepatic Homogenate (Meeting Abstract).**

(Eng.) Oyasu, R. (Northwestern Univ. Medical Sch., Chicago, IL 60611) Tomikawa, M.; Iwasaki, T.; McNamara, A. *Proc Am Assoc Cancer Res* 18: 219; 1977. (no refs.)

77-3077 Nitroxyl Free Radical Intermediate in Rat Mammary Peroxidase Catalyzed Oxidation of the Carcinogen N-Hydroxy-2-acetylaminofluorene (Meeting Abstract). (Eng.) Reigh, D. L. (Biomembrane Res. Lab., Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104) Stuart, M.; Floyd, R. A. *Biophys J* 17(2): 168; 1977. (no refs.)

77-3078 Induction of Differentiation in Mouse Neuroblastoma Cells by Hexamethylene Bisacetamide. (Eng.) Palfrey, C. (Dept. Neurobiology, Weizmann Inst. Science, Rehovot, Israel) Kimhi, Y.; Littauer, U. Z.; Reuben, R. C.; Marks, P. A. *Biochem Biophys Res Commun* 76(3): 937-942; 1977.

The effects of the potent inducer of erythroid differentiation in murine erythroleukemic cells, hexamethylene bisacetamide (HMBA), were studied in murine neuroblastoma NIE-115 cells during exponential growth. A concentration of 5 mM HMBA inhibited cell growth within 3-4 days, with almost all the cells developing neurites. An extensive network of neurites was rapidly established. With 2 mM HMBA, the culture was found to consist of a mixed population of round and neurite-bearing cells. The neurites formed in the presence of HMBA were thicker and more branched than those formed in the presence of 280 mM dimethylsulfoxide. (18 refs.)

77-3079 Specific Alkylation and Necrosis of Pulmonary Nonciliated Bronchiolar Cells by the Lung-Toxic Furan, 4-Ipomeanol (Meeting Abstract). (Eng.) Boyd, M. R. (Clinical Pharmacology Branch, NCI, NIH, Bethesda, MD 20014) *Proc Am Assoc Cancer Res* 18: 246; 1977. (no refs.)

77-3080 Effects of the Carcinogens N-[4-(5-nitro-2-Furyl)-2-Thiazolyl]acetamide (NFTA) and N-[4-(5-Nitro-2-Furyl)-2-Thiazolyl]formamide (FANFT) and Their Non-nitro Analogues on Antibody-Mediated (AMI) and Cell-Mediated (CMI) Murine Immunity (Meeting Abstract). (Eng.) Headley, D. B. (Dept. Human Oncolo-

gy, Univ. Wisconsin Medical Sch., Madison, WI 53706) Klopp, R. G.; Ewenko, J. M.; Bryan, G. T. *Proc Am Assoc Cancer Res* 18: 243; 1977. (no refs.)

77-3081 Effect of Phenothiazine, 5-DI-O-Acetyl-D-Glucosaccharo-(1-4)(6-3)-Dilacton (SLA), Caffeine, Cysteamine and Dexamethasone on the Induction of Bladder Cancer in Rats by N-[4-(5-Nitro-2-Furyl)-Thiazolyl]formamide (FANFT) (Meeting Abstract). (Eng.) Wang, C. Y. (Div. Clinical Oncology, Univ. Wisconsin Center Health Sciences, Madison, WI 53706) Hayashida, S. *Proc Am Assoc Cancer Res* 18: 100; 1977. (no refs.)

77-3082 Mutagenicity of Urine of Various Species Fed N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT) or 2-Amino-4-(5-nitro-2-furyl)thiazole (ANFT) (Meeting Abstract). (Eng.) Wang, C. Y. (Dept. Human Oncology, Univ. Wisconsin Center for Health Sciences, Madison, WI 53706) Hayashida, S.; Bryan, G. T. *Fed Proc* 36(3): 304; 1977. (no refs.)

77-3083 RNase- and ATPase-Deficient Foci in Preneoplastic Rat Liver (Meeting Abstract). (Eng.) Daoust, R. (Institut du Cancer de Montreal, Centre Hospitalier Notre-Dame, and Dept. d'Anatomie, Universite de Montreal, Montreal, Canada) *Proc Am Assoc Cancer Res* 18: 106; 1977. (no refs.)

77-3084 Evidence for a Protein Receptor of Activated Azocarcinogen (Meeting Abstract). (Eng.) Mainigi, K. D. (Inst. Cancer Res., Philadelphia, PA 19111) Sorof, S. *Proc Am Assoc Cancer Res* 18: 76; 1977. (no refs.)

77-3085 Effects of Hypocholesteremic agents on the Induction of Intestinal Tumors in Rats by Azoxymethane: Comparison of Cholestyramine and Candicidin (Meeting Abstract). (Eng.) Campbell, R. L. (Dept. Surgery, Wayne State Univ. Sch. Medicine, Detroit, MI 48201) Nigro, N. D. *Proc Am Assoc Cancer Res* 18: 98; 1977. (no refs.)

77-3086 In Vivo and In Vitro Metabolism of the Colon Carcinogen Azoxymethane (AOM) (Meeting



**Abstract).** (Eng.) Fiala, E. S. (Naylor Dana Inst., American Health Foundation, Valhalla, NY 10595) Kulakis, C.; Christiansen, G.; Weisburger, J. H. *Proc Am Assoc Cancer Res* 18: 105; 1977. (no refs.)

**77-3087 Distribution of Proliferating Cells in an Experimental Colon Carcinoma (Meeting Abstract).** (Eng.) Maskens, A. P. (Cancer Res. Unit, Clinique Saint-Michel, Brussels, Belgium) *Proc Am Assoc Cancer Res* 18: 81; 1977. (no refs.)

**77-3088 Inhibitory Effects of Selenium on 1,2-Dimethylhydrazine and Methylazoxymethanol Acetate Induction of Colon Tumors.** (Eng.) Jacobs, M. M. (Dept. Biochemistry, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., 6723 Bentner Ave., Houston, TX 77030) Jansson, B.; Griffin, A. C. *Cancer Lett (Amsterdam)* 2(3): 133-138; 1977.

The inhibitory effects of selenium (sodium selenite) on 1,2-dimethylhydrazine (DMH) and methylazoxymethanol acetate (MAM) induction of colon tumors in rats were examined. Sprague-Dawley rats received weekly dosages of either DMH (20 mg/g body wt) or MAM (20 mg/g body wt) for 18 wk. Selenium-supplemented (4 ppm) drinking water was available 1 wk prior to and throughout carcinogen administration. The addition of selenium had no effect upon wt gain; it significantly increased the number of normal-appearing colons from 4 to 14 in groups of 15 each, and it significantly decreased the number of rats developing DMH-induced tumors from 13/15 to 6/15, respectively. Selenium had no effect on the rats receiving MAM. Selenium also reduced the total number of tumors induced in DMH- (39 to 11) and MAM- (73 to 42) treated animals. With both DMH and MAM, a higher frequency of tumors was noted in the transverse section than the proximal or distal colon. (12 refs.)

**77-3089 Effect of Hepatotoxic or Nephrotoxic Agents on the Induction of Colon Cancers in Rats by 1,2-Dimethylhydrazine.** (Eng.) Fukushima, S. (Dept. Pathology, Sch. Medical Technology and Nursing, Nagoya Hoken-Eisei Univ., Dengakugakubo, Kutsukake-cho, Toyooka, Aichi 470 11, Japan) Hibino, T.; Shibata, M.; Murasaki, G.; Ogiso, T.; Ito, N. *Toxicol Appl Pharmacol* 40(3): 561-570; 1977.

The effects of liver injury induced by p,p'-diaminodiphenylmethane (DDPM) and of kidney injury induced by N-(3,5-dichlorophenyl) succinimide (NDPS) on 1,2-dimethylhydrazine (DMH) colon carcinogenesis were examined in male Wistar rats. The rats were fed diets containing 1,000 ppm DDPM (Group 1) or 5,000 ppm NDPS

(Group 2) for 8 wk, and they then received weekly sc injections of 10 mg/kg DMH for 12 wk. A third group of rats was given DMH without pretreatment. Prior administration of either DDPM or NDPS before DMH injection had no significant effect on tumor incidence, which was 54.5% (12/22 animals) in Group 1, 36.0% (9/25) in Group 2, and 45.5% (10/22) in Group 3. However, differences were noted in histological pattern, tumor size, and extent of invasion. Seven of 14 tumors in Group 1 animals were tubular adenocarcinomas, but 9/14 tumors in Group 2 rats and 8/10 in Group 3 rats were signet-ring cell carcinomas or mucinous adenocarcinomas. Tumors that developed in Group 3 rats were larger than those in rats of Groups 1 or 2. More than one-half the tumors in Groups 1 and 2 did not infiltrate beyond the submucosa; most tumors in Group 3 rats invaded the muscle layer in the serosa and metastasized to the regional lymph nodes. Possible reasons for these differences are discussed. (28 refs.)

**77-3090 Roentgenologic Demonstration of Tumors of the Colon and Rectum in the European Field Hamster (*Cricetus Cricetus* L.) (Meeting Abstract).** (Ger.) Green, U. (Abt. f. exp. Pathologie, Medizinische Hochschule Hannover, D-3000 Hannover 61, W. Germany) Reznik, G.; Eekel, H.; Rippel, W. *Z Versuchstierk* 19(1-2): 103; 1977. (no refs.)

**77-3091 Induction of DNA Repair in Human Leukocytes by Fecal Metabolites of Dimethylhydrazine (Meeting Abstract).** (Eng.) Miller, C. T. (Health Protection Branch, Ottawa, Canada) Ruddick, J. A. *Toxicol Appl Pharmacol* 41(1): 158; 1977. (no refs.)

**77-3092 The Putative Role of Dietary Fiber in DMH-Induced Colon Carcinogenesis (Meeting Abstract).** (Eng.) Barbolt, T. (Inst. Comparative and Human Toxicology, Albany Medical Coll., Albany, NY) Ringwood, N.; Abraham, R. *Toxicol Appl Pharmacol* 41(1): 157; 1977. (no refs.)

**77-3093 Mutagenicity of Benzidine and Related Compounds Employed in the Detection of Hemoglobin.** (Eng.) Ferretti, J. J. (Dept. Microbiology and Immunology, Univ. Oklahoma Health Sciences Center, Oklahoma City, OK 73190) Lu, W.; Liu, M. B. *Am J Clin Pathol* 67(6): 526-527; 1977.

Seven compounds commonly used as chromogens for the de-

tection of Hb and its derivatives were assayed for mutagenicity using the Salmonella/mammalian microsome test. Three of the compounds, benzidine, o-dianisidine, and o-tolidine, were mutagenic. Benzidine and o-tolidine are known carcinogens; therefore, there is a high probability that o-dianisidine will also prove to be carcinogenic. The four other compounds tested, o-anisidine, diphenylamine, guaicol, and o-toluidine, appeared to have no mutagenic activity. Since the three compounds with mutagenic activity have been used extensively by technologists and researchers in clinical chemistry laboratories, a potential hazard to these workers exists, and reevaluation of the use of Hb chromagens is suggested. (13 refs.)

- 77-3094 **Hexachlorobenzene Induction of 2,4-Diaminoanisole Mutagenicity In Vitro.** (Eng.) Dybing, E. (Dept. Environmental Toxicology, Natl. Inst. Public Health, Oslo 1, Norway) Aune, T. *Acta Pharmacol Toxicol (Kbh)* 40(5): 575-583; 1977.

The effects of ip injections of hexachlorobenzene (HCB) in rats on 2,4-diaminoanisole (DAA) mutagenicity in vitro were compared with those of HCB on microsomal ethylmorphine N-demethylase (EMD), a typical cytochrome P-450-mediated oxidation reaction. There was an increase in liver microsomal DAA activation to a mutagen after a dose of 10 mg/kg ip and an increase in EMD after a dose of 50 mg/kg ip. DAA mutagenicity was increased 24 hr after HCB pretreatment, but EMD was increased after 48 hr. HCB pretreatment also led to increased DAA mutagenicity in rat kidney preparations, but not in the lungs or in fetal liver. There was a sex difference in the inducing effects of HCB on EMD (stronger inductive effect in females) but not on DAA mutagenicity. It is concluded that the effect of HCB pretreatment shows similarities to the inductive effects of polycyclic aromatic hydrocarbons on DAA mutagenicity in that much higher revertant rates are seen after pretreatment with polycyclic aromatic hydrocarbons than with phenobarbital. However, there are differences in the inducing effects of HCB, polycyclic aromatic hydrocarbons, and polychlorinated biphenyls. (24 refs.)

- 77-3095 **The Transplantability and Growth of the Walker 256 Carcinosarcoma in Rats Exposed to a Polychlorinated Biphenyl, Aroclor 1254 (Meeting Abstract).** (Eng.) Kerkvliet, N. I. (Oregon State Univ., Corvallis, OR 97331) *Diss Abstr Int [B]* 37(9): 4387; 1977. (no refs.)

- 77-3096 **Differences in the Activation of  $^{14}\text{C}$ -2,2'-Di-(DCB) and  $^{14}\text{C}$ -2,4,5,2',4',5'-Hexachlorobiphenyl (HCB) to Irreversible Protein Bound Meta-**

bolites Catalyzed by Rat Liver Microsomes (Meeting Abstract). (Eng.) Hesse, S. (Dept. Toxicology, Gesellschaft für Strahlenu. Umweltforschung, D-8042 Neuherberg, W. Germany) Mezger, M. *Archiv für Pharmakologie (Berlin)* 297(11, Suppl): R22; 1977. (no refs.)

- 77-3097 **Hemangiosarcomatosis After Professional Exposure to Polyvinyl Chloride.** (Ger.) Wallnofer, H. (Medizinische Abteilung des Landeskrankenhauses, A-4840 Vocklabruck, W. Germany) Zinnagl, N. *Med Klin* 72(10): 410-413; 1977.

The case of a man who developed multiocular hemangiosarcomatosis after a relatively short and intermittent exposure to polyvinyl chloride is reported. The latent stage lasted 15 yr. The vessel areas affected were the aorta abdominalis, the intrarenal kidney vessels, and the intracerebral vessel systems. Symptomatically, the disease presented as a progressive peripheral circulatory disturbance. The possibility of previous sclerotic damage caused by carbon sulfide is discussed. (4 refs.)

- 77-3098 **Biological Effects of Vinyl Chloride: An Experimental Study.** (Eng.) Winell, M. (Section Occupational Toxicology, Dept. Occupational Medicine, Natl. Board Occupational Safety and Health, S-100 26 Stockholm, Sweden) Holmberg, B.; Kronevi, T. *Environ Health Perspect* 17: 211-216; 1976.

Liver damage caused by the exposure of albino NMRI mice to atmospheric vinyl chloride (VC) was assessed by estimating the plasma activities of alkaline phosphatase (AP), the transaminases, and lactate dehydrogenase (LDH). Three groups of 24 mice each were exposed by inhalation for 6 hr/day, 5 days/wk, to 50 ppm (52 wk) or 500 ppm VC (26 wk), or to air only (controls). The animals were also autopsied, and the tissue pathology was studied. Liver damage was indicated by a significant increase in total LDH levels after about 40 wk. After 46 wk, total LDH levels were increased about 2.5-fold following exposure to 500 ppm. A significant shift in the LDH isoenzyme profile to the M form also occurred. There was no corresponding elevation in transaminase activities, which might have served as an alternative indication of liver injury. AP activities also increased after about 40 wk: at this time levels were elevated 30%-40% and 50%-60% following exposure to 50 and 500 ppm VC, respectively. This elevation could indicate lesions in the hepatobiliary tract. Upon autopsy 12 mo after the start of exposure, no control mice had any lung adenomas or hemangiosarcomas, and 24 mice exposed to 24 ppm VC had lung adenomas and 8 had hemangiosarcomas. (30 refs.)



- 77-3099 **Light and Electron Microscopy of Pulmonary Tumors Induced in Mouse Lung by Vinyl Chloride Monomer (Meeting Abstract).** (Eng.) Suzuki, Y. (Mount Sinai Sch. Medicine, City Univ. New York, New York, NY) Selikoff, I. J. *Am J Pathol* 86(2): 24a; 1977. (no refs.)

- 77-3100 **Occupational Chemical Carcinogenesis: New Facts, Priorities and Perspectives.** (Eng.) Maltoni, C. In: *Environmental Pollution and Carcinogenic Risks*. (Lyon: International Agency for Research on Cancer): IARC Scientific Publications No. 13, INSERM Symposia Series vol. 52, pp. 127-149; 1976.

The results of experiments on the carcinogenicity of vinyl chloride (VC) are presented in 18 tables as part of a paper calling for a more active approach to the problem of environmental oncogenesis. VC was inhaled at doses from 30,000 ppm to 1 ppm for 4 hr/day, 5 days/wk for 52 wk by rats (Sprague-Dawley, Webster), mice (Swiss), and hamsters (Chinese); 50 ppm was the dividing dose in carcinogenic potential, and a variety of cancers resulted from the higher dosages. VC was also administered by inhalation at 10,000 ppm and 6,000 ppm, 4 hr/day for 1 wk. The resulting cancers were monitored in the animals and their offsprings. In addition, VC was ingested at 50.00, 16.65, and 3.33 mg/g body weight, once daily, 4-5 days/wk for 52 wk; liver angiosarcomas were the predominant resulting neoplasms. Lower dosages of 1, 0.3, and 0.03 mg/g body weight produced no tumors. Subcutaneous administration into rats of 30 mg of compound in 1 cc of water of chromite, neochromium, chromium allumen, lead chromate, molybdenum orange, cadmium sulfide, iron oxide, zinc chromate, and titanium oxide resulted in rhabdomyosarcomas and fibrosarcomas in all cases but chromite, iron oxide (red), titanium oxide, and zinc chromate. Adriamycin was tested in rats by sc injection of 2 mg in 1 cc olive oil. Tumors resulted in 35% of females and 30% of males after an average latency of 31-32 wk. Plans of experiments to test the oncological effectiveness of styrene, vinylidene chloride, and acrylonitrile are also given in tabular form. (14 refs.)

- 77-3101 **Mutagenicity of Pesticides Containing 1,3-Dichloropropene.** (Eng.) De Lorenzo, F. (I and II Cattedra di Chimica Biologica, Univ. Naples, Via Sergio Pansini 5, 80131 Naples, Italy) Degl'Innocenti, S.; Ruocco, A.; Silengo, L.; Cortese, R. *Cancer Res* 37(6): 1915-1917; 1977.

The widely used pesticides Telone and D.D. soil fumigant were tested for mutagenicity in *Salmonella typhimurium* strains TA1978, TA1535, TA100, TA1537, and TA98. D.D. soil fumigant is composed of 40% 1,3-dichloropropene, 40% 1,2-dichloropropane, and 20% other unknown chemicals.

Telone is composed of 30% cis-1,3-dichloropropene, 30% trans-1,3-dichloropropene, 20% 1,2-dichloropropane, 5% 2,3-dichloro-1-propene, 2% allyl chloride, and about 15% unknown chemicals. Both pesticides were mutagenic in strains TA100 and TA1535 in the presence and absence of a rat liver activating system; both pesticides were mutagenic in strain TA1978 only in the presence of such a system. The cis and trans isomers of 1,3-dichloropropene were strongly mutagenic in strains TA1535 and TA100, which are sensitive to base-pair substitutions, and only weakly mutagenic in strain TA1978. 2,3-Dichloro-1-propene showed similar behavior. 1,2-Dichloropropane was mutagenic in strains TA1535 and TA100 but only at concentrations 500 times higher than those of dichloropropene. The mutagenic properties of the purified components do not fully account for the mutagenicity of the commercial preparations; the difference might be due to the presence of unknown components. (12 refs.)

- 77-3102 **Metabolic Activation of Haloalkanes and Tests In Vitro for Mutagenicity.** (Eng.) Uehleke, H. (Dept. Toxicology, German Health Office, D-1 Berlin 33, Postfach, W. Germany) Werner, T.; Griem, H.; Kramer, M. *Xenobiotica* 7(7): 393-400; 1977.

The relationship between the covalent binding of metabolically activated haloalkanes and their mutagenic activity was investigated. During incubation of <sup>14</sup>C-labeled carbon tetrachloride, chloroform, halothane, and trichlorofluoromethane with liver microsomes and NADPH, considerable radioactivity was bound irreversibly to endoplasmic protein and lipid. However, no <sup>14</sup>C was detected in the ribosomal RNA. None of the four haloalkanes induced mutations after incubation with liver microsomes and the bacterial tester strains *Salmonella typhimurium* TA1535 and TA1538. Very low or no activity was associated with soluble protein or RNA when they were added to the haloalkanes-microsome incubation mixture. (54 refs.)

- 77-3103 **The Contribution of Metronidazole and Two Metabolites to the Mutagenic Activity Detected in Urine of Treated Humans and Mice.** (Eng.) Connor, T. H. (Div. Environmental Toxicology and Epidemiology, Dept. Preventive Medicine and Community Health, Univ. Texas Medical Branch, Galveston, TX 77550) Stoekel, M.; Evrard, J.; Legator, M. S. *Cancer Res* 37(2): 629-633; 1977.

The urine of two patients receiving therapeutic doses of the trichomonacide metronidazole was analyzed for mutagenic activity using the histidine auxotroph TA1535 of *Salmonella typhimurium*. The activity in the urine was significantly higher than could be accounted for by the presence of the drug. Chromatographic analysis of the urine indicated the presence

of the metabolite 1-(2-hydroxyethyl)-2-hydroxymethyl-5-nitroimidazole, which, when tested in vitro with TA1535, was 10 times more active than metronidazole. An additional urinary metabolite, 1-acetic acid-2-methyl-5-nitroimidazole, was inactive when tested similarly. The in vitro mutagenic activity of metronidazole and the two metabolites was unchanged by the addition of phenobarbital- or Aroclor-induced rat liver homogenates to the test system. In addition, metronidazole and the hydroxymethyl metabolite reverted *S. typhimurium* TA100 but not TA1537, TA1538, or TA98; the acetic acid metabolite failed to revert any of the tester strains. In studies with mice, metronidazole was required in excess of the human dose in order for significant amounts of the hydroxymethyl metabolite to be detected in the urine. Urine from mice pretreated with the hepatotoxin carbon tetrachloride prior to the administration of metronidazole demonstrated approx a 50% reduction in mutagenic activity. The formation of the urinary metabolites was inhibited. These findings indicate metabolite production from the parent compound by the liver of the intact animal that could not be determined with the standard in vitro liver homogenate system. (12 refs.)

**77-3104 On the Nature of Interaction Between the Mutagenic Drug Proflavine and the Nucleotide Guanosine 5'-Phosphate (Meeting Abstract).** (Eng.) Badea, M. G. (Univ. Tennessee, TN) *Diss Abstr Int [B]* 37(8): 3732; 1977. (no refs.)

**77-3105 Non-random Distribution of Cyclophosphamide-induced Chromosome Breaks.** (Eng.) Morad, M. (Mabarrah Hosp., Assiut, Egypt) El Zawahri, M. *Mutat Res* 42(1): 125-130; 1977.

The mutagenic effect of cyclophosphamide (CP) on human lymphocytes was studied in vivo and in vitro by QM banding of the Giemsa-stained cells. In normal lymphocytes incubated with CP for 72 hr, the incidence of chromatid breaks was 2%, 5.33%, and 6% at concentrations of 10, 100, and 1,200  $\mu\text{g}/\text{ml}$  culture media, respectively. In cells from seven patients with recurrent carcinoma of the uterus or ovary, the number of chromatid breaks and interchanges was significantly increased at 3 and 24 hr after CP therapy (2.0 g ip). All patients had previously received x-ray therapy. The chromosome aberrations were not distributed at random, since there was a significant increase in the number of breaks on chromosome 15. The break points affected the weakly fluorescent region on the chromosomes more than the strongly fluorescent region. (16 refs.)

**77-3106 Study on Cytological Effects of Carofur--A New Mutagen.** (Eng.) Vig, B. K. (Dept. Biology,

Univ. Nevada, Reno, NV 89507) Natarajan, A. T.; Zimmermann, F. K. *Mutat Res* 42(1): 109-116; 1977.

Chinese hamster cells (line V-79) and human WBC, and CBA mice were treated with the antibacterial agent carofur (nifurprazinium). At concentrations as low as 20 ppm, carofur induced deletion-type chromosome aberrations in Chinese hamster cells and human WBC. This effect resembles that of fluorodeoxyuridine, but it may not necessarily involve the same basic mechanism. When Chinese hamster cells were treated with  $\geq 5$  ppm carofur for 24 hr, a threefold increase in the frequency of somatic sister chromatid exchanges occurred. An interesting phenomenon, centromeric association, was observed in the bone marrow cells of mice treated with carofur, in which the centromeres of the acrocentric chromosomes were oriented toward each other in groups of two or more. (5 refs.)

**77-3107 Response of Hematopoietic Cell Lines Derived from Patients with Down's Syndrome and from Normal Individuals to Mitomycin C and Caffeine.** (Eng.) Banerjee, A. (Dept. Experimental Biology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263) Jung, O.; Huang, C. C. *J Natl Cancer Inst* 59(1): 37-39; 1977.

Five hematopoietic cell lines from patients with Down's syndrome (DS) and three from normal subjects were treated with mitomycin C (MC, 0.1-5  $\mu\text{g}/\text{ml}$ ) for 6-26 hr and with caffeine (CAF, 250-1,000  $\mu\text{g}/\text{ml}$ ) for 6-48 hr. MC and CAF induced chromosome aberrations in both normal and DS cultures, and the extent of the aberrations was correlated with dose level and time. The sensitivity of the two groups of hematopoietic cell lines to treatment did not differ significantly. MC severely reduced cell viability in both DS and normal cultures. However, inhibition of mitosis by MC was somewhat stronger in the DS lines than in the normal lines. (24 refs.)

**77-3108 The Effect of Caffeine on the Survival of HeLa S3 Cells as a Function of X-Ray Dose and Cellular Age (Meeting Abstract).** (Eng.) Busse, P. M. (Dept. Anatomy, Washington Univ. Sch. Medicine, St. Louis, MO 63110) Bose, S. K.; Jones, R. W.; Tolmach, L. J. *Biophys J* 17(2): 245; 1977. (no refs.)

**77-3109 Inhibition of Initiation of HeLa Cell Replicons by Methyl Methanesulfonate.** (Eng.) Painter, R. B. (Lab. Radiobiology, Univ. California at San Francisco, San Francisco, CA 94143) *Mutat Res* 42(2): 299-304; 1977.

Methyl methanesulfonate (MMS  $5 \times 10^{-4}$  or  $10^{-3}$  M) in the



culture medium inhibited the rate of DNA synthesis in HeLa cells in a dose-dependent manner. By using short (5-min) incubations with  $^3\text{H}$ -thymidine and analyzing the newly made DNA by velocity sedimentation on alkaline sucrose gradients, it was found that the first effect of MMS on DNA replication, at 0.5 hr after treatment, was inhibition of replication initiation. Recovery from this effect seemed to begin by 2 3/4 hr after treatment. The second effect of MMS, which was evident at 2 hr after treatment, was to slow or block chain elongation. The results support the concept that MMS produces x-ray-like lesions in mammalian DNA. (12 refs.)

- 77-3110 **Chromosome Aberrations and Dominant Lethality of Mouse Embryos after Paternal Treatment with Triethylenemelamine.** (Eng.) Hitotsumachi, S. (Central Res. Div., Takeda Chemical Industries Ltd., Osaka, Japan) Kikuchi, Y. *Mutat Res* 42(1): 117-124; 1977.

Adult male CF#1 mice were inoculated ip with 0.3 mg/kg triethylenemelamine (TEM) and then mated with untreated females twice a week over the next 24 days. On day 3 of gestation, embryos were flushed out from the uteri and examined cytologically and cytogenetically. A dominant-lethal test was conducted using males treated in the same way. Paternal treatment of TEM caused developmental retardation of the embryos obtained from matings on postinjection day 20. These embryos frequently showed micronuclei in interphase cells and structural chromosome aberrations in metaphases. Most of these aberrations were chromosome types, such as breaks and exchanges and premature chromosome condensations. The developmental retardation and the frequency of chromosome aberrations were most marked in the matings on postinjection day 13. Similarly, the highest dominant-lethal effect was shown in the group mated on postinjection days 11-13. It is concluded that TEM induces chromosome damage in postmeiotic germ cells and that this damage in turn produces chromosome aberrations in the embryos, resulting in a high incidence of dominant lethality. (12 refs.)

- 77-3111 **Effect and Metabolism of Ethionine in Hamster (Meeting Abstract).** (Eng.) Brada, Z. (Papanicolaou Cancer Res. Inst. Miami, FL 33123) Bulba, S.; Altman, N. H. *Proc Am Assoc Cancer Res* 18: 239; 1977. (no refs.)

- 77-3112 **Mutagenicity and Possible Carcinogenicity of Hair Colourants and Constituents.** (Eng.) Venitt, S.; Searle, C. E. In: *Environmental Pollution and Carcinogenic Risks*. (Lyon: International Agency for Research on Cancer): IARC Scientific Publications No. 13, INSERM Symposia Series, Vol. 52, pp. 263-272; 1976.

Acute myeloid leukemia developed in a middle-aged woman who had used two semi-permanent hair colorants (GS, which contains 2-nitro-p-phenylenediamine (2NPPD) and 4-nitro-o-phenylenediamine (4NOPD); and RB, which contains CL Acid Black 107 and 4 amino-2-nitrophenol. To investigate a possible carcinogenic connection, mice (DBA/1 and A strains) received twice weekly applications of each colorant to the clipped dorsal skin: strain A, 0.4 ml and DBA/1, 0.2 ml per application. Early development of lymphoid tumors was significant in both strains and with both dyes; GS-treated DBA/1 mice also developed genital tract sarcomas. Bacterial mutation studies were then undertaken using *Salmonella typhimurium* TA 1535 and TA 1538. GS and RB and other colorants (0.1 ml samples) were assayed with or without the addition of a supernatant fraction from rat liver combined with an NADPH generating system (S-9 mix). Positive results were obtained in TA 1538, and all colorants were mutagenic, especially in the absence of S-9 mix, although RB differed in needing the S-9 mix to show mutagenic activity. A number of permanent colorants for domestic and professional use were then assayed under the same conditions. The compounds included phenylenediamines, diaminotoluenes, diaminoanisoles, and aminophenols. None of the colorants was mutagenic in the absence of metabolic activation, and they were much less active when tested without prior oxidation. (19 refs.)

- 77-3113 **In Vitro Acylation of the  $\epsilon$ -Amino Group of L-Lysine in Calf Thymus Histones by the Carcinogen,  $\beta$ -Propiolactone.** (Eng.) Segal, A. (Lab. Organic Chemistry Carcinogenesis, Inst. Environmental Medicine, New York Univ. Sch. Medicine, New York, NY 10016) *Chem Biol Interact* 15(4): 319-326; 1976.

The in vitro acylation of the  $\epsilon$ -amino group of L-lysine in calf thymus histones by  $\beta$ -propiolactone (BPL) is investigated. The synthesis of  $\epsilon$ -N-(3-hydroxypropionyl)lysine (HPL) and  $\epsilon$ -N-(2-carboxyethyl)lysine (CEL) from BPL and L-lysine involved the protection of the  $\alpha$ -amino group of the L-lysine through the formation of a copper chelate. The infrared spectrum of HPL contained a band at  $1650\text{ cm}^{-1}$  (amide carbonyl) not present in the spectrum of CEL. HPL contained one titratable acidic group and one basic group, while CEL contained two titratable acidic groups and two basic groups. HPL and CEL gave single spots on silica gel thin layer chromatography analysis. The elution profiles after chromatography on cation-exchange resin of the trypsin-pronase digests of control were determined. The profiles were very similar except for the appearance of a ninhydrin-positive peak in fractions 44-49. When HPL and CEL were added to the trypsin-pronase digest of control calf thymus histones and the mixture was chromatographed, both compounds were resolved, HPL in fractions 44-49 and CEL in fractions 116-118. When HPL and CEL were applied to the cation exchange column separately or together, they were eluted in the same fractions as

when both compounds were chromatographed with the trypsin-pronase digest of control calf thymus histones. Compound CEL was not identified in the trypsin-pronase digest of BPL-alkylated calf thymus histones. All chromatographic runs were performed in duplicate. Additional support for the BPL acylation of the  $\epsilon$ -amino group in L-lysine was the decrease in ninhydrin binding capacity (50%) of the trypsin-pronase digest of calf thymus histones following BPL reaction compared to the digest of control calf thymus histones. The absorbance for the trypsin-pronase digest of BPL- reacted calf thymus histones was 0.401, and for the trypsin-pronase digest of control calf thymus histones the absorbance was 0.800. The results may be of potential significance in chemical carcinogenesis. (25 refs.)

**77-3114 Isolation and Cultivation of Cells from Carageenan-Induced Granulomas of the Mouse (Meeting Abstract).** (Eng.) Bonney, R. J. (Merck Inst. for Therapeutic Res., Rahway, NY 07065) Dahlgren, M. E.; Davies, P. *In Vitro* 13(3): 174-175; 1977. (no refs.)

**77-3115 Mammary Tumorigenesis and Pathologic Changes of the Reproductive Tract of Female Mice Continuously Fed Diethylstilbestrol and 17 $\beta$ -Estradiol for 18 Months (Meeting Abstract).** (Eng.) Norvell, M. J. (NCTR, Jefferson, AR) Farmer, J. H.; Highman, B.; Shellenberger, T. E. *Proc Am Assoc Cancer Res* 18: 243; 1977. (no refs.)

**77-3116 Radioimmunologic Research on Diethylstilbestrol in Plasma and Tissues of Young Cattle (Meeting Abstract).** (Fre.) Pantaleon, J. ((No affiliation given)) Richou-Bac, L.; Mollet, M. F.; Boursier, B.; Cumont, G. *Sem Hop Paris* 53(11/12): 710; 1977. (no refs.)

**77-3117 Lack of Influence of Hypophysectomy on Estrogen-induced DNA Synthesis in Leydig Cells of BALB/c Mice.** (Eng.) Huseby, R. A. (Div. Oncology, Henry Ford Hosp., Detroit, MI 48202) Samuels, L. T. *J Natl Cancer Inst* 58(4): 1047-1049; 1977.

In mice susceptible to Leydig cell tumor induction, estrogen treatment induces a spurt of DNA synthesis within the first few days; this synthetic activity generally subsides until areas of Leydig cell hyperplasia develop several months later. Autoradiographic and quantitative biochemical studies of BALB/c mice implanted with 10-mg pellets of 10% diethyl-

stilbestrol showed that the initial DNA synthetic activity occurred in the absence of hypophysis and apparently was the result of direct effects of the hormone on Leydig cells. Although hypophysectomy inhibited sperm maturation,  $^3\text{H}$ -thymidine incorporation into spermatogonia was reduced only slightly 2 wk after surgery, as was the induced DNA spurt in the interstitial tissues. (11 refs.)

**77-3118 Exogenous Estrogens and Endometrial Carcinoma.** (Eng.) Reeves, K. O. (Dept. Obstetrics and Gynecology, Baylor Coll. Medicine, Houston, TX) Kaufman, R. H. *J Reprod* 18(6): 297-300; 1977.

A review is presented of eight cases of severe, atypical endometrial changes in women, aged 37-45 yr, who had taken the oral contraceptive Oracon for 4-8 yr. Oracon, now withdrawn from the market, is a sequential contraceptive containing 100  $\mu\text{g}$  of ethinyl estradiol and 25 mg of dimethisterone per tablet. The endometria of seven of the patients demonstrated well-differentiated adenocarcinomas; that of the other patient showed severe atypical endometrial hyperplasia. Various degrees of a clear-cell pattern of adenocarcinoma, with a clear, translucent cytoplasm, were found in four patients. In one, squamous metaplasia was observed. It is recommended that close attention be given to women who have used Oracon. (20 refs.)

**77-3119 Steroid Contraceptive Use and Cervical Dysplasia: Increased Risk of Progression.** (Eng.) Stern, E. (Div. Epidemiology, Sch. Public Health, Univ. California, Los Angeles, CA 90024) Forsythe, A. B.; Youkeles, L.; Coffelt, C. F. *Science* 196(4297): 1460-1462; 1977.

A prospective study was made of the effects of steroid contraceptive pill usage in 300 women found during routine screening from 1967 to 1971 in Los Angeles County to have dysplasia of the cervix, a precursor of cancer in situ. The influence of the pill on the progression of dysplasia was studied for periods of up to 86 mo. Among nonusers there was a trend of progressive decrease in the severity of dysplasia (reversal) from the time of initial observation. The extent of reversal tended to be greater among pill users over the first year, but thereafter pill users tended to demonstrate more dysplasia than the nonusers. Thus, there was a biphasic difference in response to the pill, beneficial over the short term but adverse over the long term. This greater degree of dysplasia among pill users was more evident when women with early reversal were excluded from the analysis. In a sample of 300 normal women not initially diagnosed with dysplasia, there was no evidence of a differential effect of pill usage on cervical morphology. It was estimated that the probability of progressing from dysplasia to cancer in situ in pill users is 0.30 at 7 yr, compared to a probability of 0.05 in nonusers. (17 refs.)



77-3120 **Mammary Hyperplasia and Neoplasia in Beagles Receiving Low Levels of Medroxyprogesterone Acetate (Meeting Abstract).** (Eng.) Fowler, E. H. (Univ. Rochester, Sch. Medicine, Rochester, NY 14642) Vaughan, T.; Gotesik, F.; Reichhart, P.; Reed, C. *Proc Am Assoc Cancer Res* 18: 212; 1977. (no refs.)

77-3121 **Role of Biogenic Amines and Hypothalamic Hormones in Control of Prolactin, TSH and GH Release and of Carcinogen-induced Mammary Tumor Growth in Rats (Meeting Abstract).** (Eng.) Chen, H. J. (Michigan State Univ., East Lansing, MI 48823) *Diss Abstr Int [B]* 37(9): 4333; 1977. (no refs.)

77-3122 **Antagonistic Activity of Poly(4-vinylpyridine-N-oxide) to the Inhibition of Viral Interferon Induction by Asbestos Fibres.** (Eng.) Hahon, N. (US Public Health Service, Appalachian Lab. Occupational Safety and Health, Morgantown, WV 26505) Booth, J. A.; Eckert, H. L. *Br J Ind Med* 34(2): 119-125; 1977.

The protection afforded by poly(4-vinylpyridine-N-oxide) (PVPNO) against the suppression, by both serpentine (Canadian and Rhodesian chrysotiles) and amphibole (amosite, crocidolite, and anthophyllite) asbestos fibers, of interferon (IF) synthesis was investigated in cell monolayers. The depressive activity of both kinds of asbestos fibers on IF induction by influenza virus was significantly diminished or abolished completely when either the fibers or Rhesus monkey kidney (LIC-MK<sub>2</sub>) cell monolayers were pretreated with PVPNO. Max antagonistic activity of the polymer was time- and concentration-dependent. Pretreating asbestos fibers with PVPNO was more rapid and effective in encouraging viral IF synthesis than pretreating the cell monolayers. Within 30 min of pretreating asbestos fibers with PVPNO, their effect on IF production was nullified; however, 16-hr pretreatment of cell monolayers had only a limited effect on the subsequent ability of asbestos fibers to suppress IF induction in these cells. Viral multiplication in the presence of asbestos fiber-treated cell monolayers attained a twofold higher level than that seen in normal monolayers or those containing polymer-pretreated asbestos fibers. These results were related to the suppression of IF production. The mechanism by which PVPNO confers protection against the activity of asbestos fibers is not yet known. (31 refs.)

77-3123 **Magnesium Versus Lead in Dietary Induction of Rat Neoplasms (Meeting Abstract).** (Eng.) McCreary, P. A. (Rush-Presbyterian-St. Luke's Medical

Center, Chicago, IL) Laing, G. H.; Coogan, P. S.; Hass, G. M. *Am J Pathol* 86(2): 26a; 1977. (no refs.)

77-3124 **Carcinogenicity of Carboxymethylcellulose in Rats (Meeting Abstract).** (Eng.) Teller, M. N. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021) Brown, G. B. *Proc Am Assoc Cancer Res* 18: 225; 1977. (no refs.)

77-3125 **Electron Microscopic Cytochemical and Morphometric Study of Some Enzyme Activities in the Mitochondria of Thyroid Cells During Malignant Transformation.** (Rus.) Dmitrieva, N. P. (Section Electron Microscopy, N. K. Kol'tsov Inst. Developmental Biology, Acad. Sciences USSR, Moscow, USSR) Stefanov, S. B.; Amirkhanian, E. A. *Biull Eksp Biol Med* 83(4): 452-455; 1977.

Random-bred albino rats were exposed to daily doses of 6-methyluracil (doses unspecified). Six to 12 mo later, most animals developed hyperplasia of the thyroid gland and, 14-20 mo later, adenocarcinomas of the thyroid gland. Animals were sacrificed 6, 8, 10, 12, 15, and 20 mo after the start of the treatment, and the mitochondrial cytochrome oxidase (CCO) and succinate dehydrogenase (SDH) content in the thyroid gland was assessed cytochemically. The mitochondrial CCO activity was slightly inhibited in glands with advanced hyperplasia (3.26 U, compared to 3.93 U in the control animals). The proportion of mitochondria showing intensive SDH activity was decreased in the advanced hyperplasia cells (9.0%) and, even more so, in the adenocarcinoma cells (3.0%) compared to the cells from control animals (60%). (7 refs.)

77-3126 **Mutation Induction in Synchronous Hamster Cells (Meeting Abstract).** (Eng.) Aebersold, P. M. (Univ. California, Berkeley, CA 94720) *Diss Abstr Int [B]* 37(9): 4313; 1977. (no refs.)

77-3127 **Epidermal Ribosome Accumulation During Two-Stage Skin Tumorigenesis.** (Eng.) De Young, L. M. (McArdle Lab. Cancer Res., Univ. Wisconsin Medical Sch., Madison, WI 53706) Argyris, T. S.; Gordon, G. B. *Cancer Res* 37(2): 388-393; 1977.

The increase, on the backs of female CD-1 mice, in interfollicular epidermal ribosomes initiated with 7,12-dimethylbenz(a)anthracene [200 nanomoles (nmol)] and promoted with 12-O-tetradecanoyl-phorbol-13-acetate, (17

nmol) was disproportionate to the increase in epidermal wet wt, protein, and DNA. Ribosome numbers were determined electron microscopically from non-detergent treated (free ribosomes) and detergent-treated (membrane bound ribosomes) postnuclear supernatant collected from the pellet at the bottom of a 2.0 M and a 1.35-2.0 M sucrose layer, respectively. Although ribosome numbers increased five to sixfold 48 hr after the first, fourth, or eighth application of 12-O-tetradecanoyl-phorbol-13-acetate, epidermal tissue increased only two- to threefold at these times. This disproportionate increase was due to the concurrent two- to threefold increase in ribosomes per g of epidermis and per mg of DNA. The tissue concentration and cellular content of ribosomes were also increased in the epidermal component of induced squamous papillomas. Other studies have demonstrated that during growth of other tissues and organs, ribosome accumulation is proportionate to the accumulation of tissue and/or cells. The results of this study indicate that the epidermis may have unique kinetics of ribosome accumulation during induced growth. Furthermore, the findings suggest the possibility that other tumor-prone surface epithelia, such as the linings of the respiratory and gastrointestinal tracts, have similar kinetics of ribosome accumulation during induced growth. (28 refs.)

**77-3128 Metabolism of 12-O-Tetradecanoylphorbol-13-Acetate (TPA) in Adult and Newborn Skin and Newborn Epidermal Cells in Culture (Meeting Abstract).** (Eng.) Berry, D. L. (Biology Div., ORNL, Oak Ridge, TN 37830) Fischer, S. M.; Slaga, T. J. *Proc Am Assoc Cancer Res* 18: 88; 1977. (no refs.)

**77-3129 Differential Effects of a Tumor Promoter on Human and Hamster Cells in Culture (Meeting Abstract).** (Eng.) O'Brien, T. G. (Wistar Inst., Philadelphia, PA 19104) *Proc Am Assoc Cancer Res* 18: 90; 1977. (no refs.)

**77-3130 Membrane Effects of Tumor-Promoting Phorbol Esters (Meeting Abstract).** (Eng.) Wenner, C. E. (Roswell Park Memorial Inst., Buffalo, NY) Moroney, J. V. *Proc Am Assoc Cancer Res* 18: 241; 1977. (no refs.)

**77-3131 Development of Liver Tumors in Rats Treated with the Peroxisomal Enzyme Inducer Nafenopin (Meeting Abstract).** (Eng.) Reddy, J. K. (Northwestern Univ. Medical Sch., Chicago, IL) Rao, M. S. *Am J Pathol* 86(2): 25a; 1977. (no refs.)

**77-3132 Detection of Chemical Carcinogens by Unscheduled DNA Synthesis in Rat Liver Primary Cell Cultures.** (Eng.) Williams, G. M. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY 10595) *Cancer Res* 37(6): 1845-1851; 1977.

Unscheduled DNA synthesis was estimated in primary rat liver cell cultures treated with various chemical procarcinogens, analogs, and noncarcinogens. Unscheduled DNA synthesis amounted to: 0-5.8 grains/nucleus (g/n: no agent added), 35.0 g/n (0.1 mM aflatoxin B<sub>1</sub>), 16.8 g/n (0.1 mM aflatoxin B<sub>2</sub>), 42.4 g/n (1 mM 2-acetylaminofluorene), 0.8 g/n (1 mM 4-acetylaminofluorene), 22.1 g/n (0.1 mM 3'-methyl-4-dimethylaminoazobenzene: 3'-MDMAB), 11.4 g/n (0.1 mM 4'-MDMAB), 7.7 g/n (0.1 mM 2-MDMAB), 25.1 g/n (0.1 mM 4-aminoazobenzene), 16.5 g/n [1 mM 7,12-dimethylbenz(a)anthracene], 6.6 g/n [1 mM benz(a)anthracene], and 3.6 g/n (1 mM anthracene). The system demonstrates a substantial sensitivity to chemical procarcinogens requiring metabolic activation, and it may be useful for identifying agents deserving consideration as potential carcinogens. (68 refs.)

**77-3133 Infidelity of DNA Synthesis: A Biochemical Screening System for Environmental Mutagens or Carcinogens (Meeting Abstract).** (Eng.) Sirover, M. A. (Inst. for Cancer Res., Fox Chase Cancer Center, Philadelphia, PA 19111) Shearman, C. W.; Loeb, L. A. *Proc Am Assoc Cancer Res* 18: 104; 1977. (no refs.)

**77-3134 The Induction of DNA Repair in Primary Rat Liver Cultures as a Screen for Chemical Carcinogens (Meeting Abstract).** (Eng.) Williams, G. M. (Naylor Dana Inst. Disease Prevention, American Health Foundation, Valhalla, NY) *Toxicol Appl Pharmacol* 41(1): 158; 1977. (no refs.)

**77-3135 Induction of Micronuclei by Carcinogens (Meeting Abstract).** (Eng.) Friedman, M. A. (Dept. Pharmacology and Biostatistics, Medical Coll. Virginia, Richmond, VA) Carter, W. H.; Staub, J.; Segreti, A. *Toxicol Appl Pharmacol* 41(1): 157-158; 1977. (no refs.)

**77-3136 Mutagen-Induced Immunoreactivity to Antinucleoside Antibodies in Human Cells (Meeting Abstract).** (Eng.) Neubort, S. (Albert Einstein Coll. Medi-



cine, Bronx, NY 10461) Blake, C.; Bases, R. *Proc Am Assoc Cancer Res* 18: 86; 1977. (no refs.)

**77-3137 The Opposite Effects of Carcinogens and Dicoumarol on the Activity of the Postmicrosomal Liver and Lung D-T Diaphorase (Meeting Abstract).** (Eng.) Schor, N. A. (Tulane Univ. Sch. Medicine, New Orleans, LA) *Am J Pathol* 86(2): 28a-29a; 1977. (no refs.)

**77-3138 Hepatic and Extrahepatic Metabolism of <sup>14</sup>C-Styrene Oxide.** (Eng.) Ryan, A. J. (Pharmacology Branch, Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC 27709) James, M. O.; Ben-Zvi, Z.; Law, F. C.; Bend, J. R. *Environ Health Perspect* 17: 135-144; 1976.

The activities of the epoxide-metabolizing enzymes glutathione S-transferase (GT) and epoxide hydrase (EH), with 8-<sup>14</sup>C-styrene oxide as substrate, were studied in subcellular fractions of liver, lungs, kidney, and intestinal mucosa of rabbits, guinea pigs, perinatal and adult rats, and rats pretreated with phenobarbital (PB), 1,2,3,4-dibenzanthracene, pregnenolone-16 $\alpha$ -carbonitrile (PCN), and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), and also in isolated perfused rat liver and rabbit lungs. The liver had the highest GT and EH activities in each species. The liver and kidney GT activities were higher in the rat and guinea pig than in the rabbit. Rat testis also had appreciable GT activity. In perinatal rats, EH and GT developed at different rates in each tissue. PB pretreatment increased both enzyme levels in the livers of rats of both sexes; PCN pretreatment induced only GT in the female liver. TCDD doubled renal EH activity and PB increased intestinal EH activity in both sexes, but the other extrahepatic EH and GT levels were unaffected by any pretreatment. When styrene oxide biotransformation was studied in isolated perfused rat liver and rabbit lung preparations, conjugation with glutathione was a major metabolic pathway, although significant amounts of diol were also formed. In rat liver, 27%-40% of the administered styrene oxide was excreted via the bile, mainly as S-(1-phenyl-2-hydroxyethyl)glutathione. (23 refs.)

**77-3139 Effect of Trichloropropene Oxide on the Ability of Polyaromatic Hydrocarbons and Their "K-Region" Oxides to Initiate Skin Tumors in Mice and to Bind to DNA In Vitro.** (Eng.) Berry, D. L. (Cancer and Toxicology Program, Biology Div., Oak Ridge Natl. Lab., Post Office Box Y, Oak Ridge, TN 37830) Slaga, T. J.; Viaje, A.; Wilson, N. M.; DiGiovanni, J.; Juchau, M. R.; Selkirk, J. K. *J Natl Cancer Inst* 58(4): 1051-1055; 1977.

The potent epoxide hydrase inhibitor 1,1,1-trichloro-2,2-propylene oxide (TCPO, 10  $\mu$ moles) enhanced the tumor-initiating ability of benzo(a)pyrene [BP, 200 nanomole (nmol)] and 3-methylcholanthrene (MC, 50 nmol), but had no effect on 9,10-dimethyl-1,2-benzanthracene (DMBA, 1 nmol) initiation in a two-stage tumorigenesis system in female Charles River CD-1 mice. The tumor-initiating ability of dibenz(a,h)anthracene (DBA, 200 nmol) was decreased by prior or topical treatment with TCPO. The tumor-latency period of BP and MC was decreased by TCPO; that of DMBA and DBA was unaffected. Topical treatment with TCPO did not initiate tumors in the two-stage system, nor did it cause any histopathologic changes in the skin. The K-region epoxide of BP, DMBA, and MC were weak tumor initiators compared with the parent compounds. TCPO only slightly increased or had no effect on the tumor initiating activity of these epoxides. Pretreatment with croton oil 18 hr prior to initiation with BP 4,5-epoxide (400 nmol) also slightly enhanced the tumorigenic response in mouse skin. DBA 5,6-epoxide, when tested as a complete carcinogen at high doses (1 mg/day for 10 days), was weak, with activity comparable to that of DBA. TCPO only slightly increased the in vitro epidermally mediated covalent binding of the parent polycyclic hydrocarbons to DNA. (27 refs.)

**77-3140 Mechanism of Microsomal Metabolism of Benzo(a)pyrene to 7,8-Diol-9,10-Epoxides: Characterization of Intermediates and Products and Interaction with DNA (Meeting Abstract).** (Eng.) Yang, S. K. (NCI, Bethesda, MD 20014) Gelboin, H. V.; Kakefuda, T. *Proc Am Assoc Cancer Res* 18: 202; 1977. (no refs.)

**77-3141 Template Activity of Calf Thymus DNA Modified by a Dihydrodiol Epoxide Derivative of Benzo(a)pyrene.** (Eng.) Leffler, S. (Inst. Cancer Res., Coll. Physicians and Surgeons, Columbia Univ., New York, NY 10032) Pulkrabek, P.; Grunberger, D.; Weinstein, I. B. *Biochemistry* 16(14): 3133-3136; 1977.

The effects of covalent binding to DNA of a reactive derivative of benzo(a)pyrene on template activity during in vitro transcription with RNA polymerase were determined. Calf thymus DNA, modified by reaction with ( $\pm$ )-7 $\beta$ ,8 $\alpha$ -dihydroxy-9 $\alpha$ ,10 $\alpha$ -epoxy-7,8,9,10-tetrahydro benzo(a)pyrene, was transcribed with *Escherichia coli* DNA-dependent RNA polymerase. With increasing levels of modification, there was a progressive inhibition of transcription. The inhibition was much greater under conditions of continuous reinitiation of transcription than under conditions in which only one RNA chain was synthesized per initiation site. This suggested that the modified sites block the movement of polymerase along the template and prevent recycling of the

enzyme. Consistent with this interpretation were analyses of RNA transcripts on sucrose density gradients that showed a progressive decrease in av RNA chain length as the extent of template modification increased. In contrast to the inhibitory effect on chain elongation, the modified DNA had an increased number of initiation sites for transcription. These results are consistent with separate physical studies indicating that modification of DNA by this benzo(a)pyrene derivative can induce small localized regions of denaturation. (28 refs.)

**77-3142 Different Catalytic Activities of Two Highly Purified Cytochrome P-450 (LM)S in the Metabolism and DNA-Binding of Benzo(a)pyrene and (-)-Trans-7,8-Dihydroxy-7,8-Dihydrobenzo(a)pyrene (Meeting Abstract).** (Eng.) Deutsch, J. (NCI, Bethesda, MD 20014) Leutz, J.; Coon, M.; Gelboin, H. *Proc Am Assoc Cancer Res* 18: 209; 1977. (no refs.)

**77-3143 Role of Microsomes and Nuclear Envelope in Benzo(a)pyrene (BP) Activation and Binding (Meeting Abstract).** (Eng.) Pezzuto, J. M. (New Jersey Medical Sch., Newark, NJ 07103) Lea, M. A.; Yang, C. S. *Proc Am Assoc Cancer Res* 18: 214; 1977. (no refs.)

**77-3144 Metabolic Oxidation of Benzo(a)pyrene (BP) by Rat Liver Nuclear Membrane (Meeting Abstract).** (Eng.) Lesko, S. A. (Johns Hopkins Univ., Baltimore, MD 21205) Ts'o, P. O. *Proc Am Assoc Cancer Res* 18: 171; 1977. (no refs.)

**77-3145 Absolute Configuration of a Ribonucleic Acid Adduct Formed In Vivo by Metabolism of Benzo(a)pyrene (Letter to Editor).** (Eng.) Nakahishi, K. (Dept. Chemistry, Columbia Univ., New York, NY 10027) Kasai, H.; Cho, H.; Harvey, R. G.; Jeffrey, A. M.; Jennette, K. W.; Weinstein, I. B. *J Am Chem Soc* 99(1): 258-260; 1977.

The experimental procedure for the isolation of the RNA adduct formed in bovine bronchial explants by metabolism of benzo[a]pyrene is presented. The absolute configuration was determined. (21 refs.)

**77-3146 Benzo(a)pyrene Binding to Chromatin Components of Calf Thymus Nuclei (Meeting Abstract).** (Eng.) Jahn, C. L. (Cornell Univ. Graduate Sch.

Medical Sciences and Memorial Sloan-Kettering Cancer Center, New York, NY 10021) Jenson, J. C.; Litman, G. W. *Proc Am Assoc Cancer Res* 18: 113; 1977. (no refs.)

**77-3147 Formation of Benzo(a)pyrene-Glucuronides in Cell Culture (Meeting Abstract).** (Eng.) Baird, W. M. (Wistar Inst., Philadelphia, PA 19104) Chern, C. J.; Diamond, L. *Proc Am Assoc Cancer Res* 18: 90; 1977. (no refs.)

**77-3148 Initiation of Mouse Skin Tumors During Prolonged Exposure to Benzo(a)pyrene (Meeting Abstract).** (Eng.) Burns, F. J. (New York Univ. Inst. Environmental Medicine, New York, NY 10016) Albert, R. E.; Pereira, M. A. *Proc Am Assoc Cancer Res* 18: 212; 1977. (no refs.)

**77-3149 Histogenesis of Squamous Metaplasia in the Tracheobronchial Epithelium of the Syrian Golden Hamster (Meeting Abstract).** (Eng.) Becci, P. (Univ. Maryland Sch. Medicine, Baltimore, MD) McDowell, E. M.; Trump, B. F. *Am J Pathol* 86(2): 65a-66a; 1977. (no refs.)

**77-3150 Determination of 1,2- and 3,4-Benzpyrene Plus Other Polycyclic Aromatic Hydrocarbons in Smoked Meat Products by Mass Fragmentography.** (Ger.) Jahr, D. (Landesuntersuchungsamt für das Gesundheitswesen Sudbayern, Fachbereich Chemie, Lothstrasse 21, D-8000 Munich 40, W. Germany) Hollerer, G. *Z Lebensm Unters Forsch* 163(1): 1-3; 1977.

Smoked meat products were analyzed for polycyclic aromatic hydrocarbons by combined gas chromatography-mass spectrometry with monitoring of two characteristic ions (mass fragmentography). The procedure shortened the analysis time and increased the sensitivity of determination of benzo-fluoranthene, 1,2-benzopyrene, and 3,4-benzopyrene. (4 refs.)

**77-3151 Proteins and Carcinogen-Protein Complexes in Mouse Mammary Gland Cytosol (Meeting Abstract).** (Eng.) Dickens, M. S. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA 19111) *Proc Am Assoc Cancer Res* 18: 77; 1977. (no refs.)



- 77-3152 **Enhancement of 5-Iododeoxyuridine-induced Endogenous C-Type Virus Activation by Polycyclic Hydrocarbons: Apparent Lack of Parallelism Between Enhancement and Carcinogenicity.** (Eng.) Yoshikura, H. (Dept. Genetics, Inst. Medical Science, Univ. Tokyo, P.O. Takanawa, Tokyo, Japan) Zajdela, F.; Perin, F.; Perin-Roussel, O.; Jacquignon, P.; Latarjet, R. *J Natl Cancer Inst* 58(4): 1035-1046; 1977.

The effect of carcinogenic chemicals on the induction of endogenous C-type virus by 5-iododeoxyuridine (IUdR) was tested in mouse MLg cells. After pretreatment with IUdR at 2.5, 5, or 10  $\mu\text{g/ml}$ , the cells were treated with 3-methylcholanthrene (MC, 1  $\mu\text{g/ml}$ ) or 7,12-dimethylbenz(a)anthracene (DMBA, 2  $\mu\text{g/ml}$ ) in the presence of microsomal enzymes and NADPH. At 5  $\mu\text{g/ml}$  IUdR, the carcinogens produced a five- to sixfold increase in virus induction; at 10  $\mu\text{g/ml}$  the increase was only twofold. The enhancing activity of MC and DMBA was dependent on the presence of the microsomal enzymes and NADPH. 7,8-Benzoflavone (2  $\mu\text{g/ml}$ ), an inhibitor of hydrocarbon metabolism in hamster embryo cultures, significantly reduced this activity. Screening experiments involving 30 polycyclic hydrocarbons showed no correlation between their ability to enhance virus activation and their in vivo carcinogenicity in the skin. (33 refs.)

- 77-3153 **The Use of Primary Rat Liver Parenchymal Cells in Evaluating Cellular Response to Toxic Metals and Carcinogenic Polycyclic Hydrocarbons (Meeting Abstract).** (Eng.) Huisinigh, J. L. (Environmental Protection Agency, Health Effects Res. Lab., Research Triangle Park, NC 27711) Inmon, J. P.; King, L. C.; Williams, K.; Waters, M. D. *In Vitro* 13(3): 182; 1977. (no refs.)

- 77-3154 **Ultrastructural Study of Formal Pathogenesis of Experimentally Induced Rhabdomyosarcomas.** (Ger.) Riede, U. N. (Pathologisches Institut der Universität Freiburg im Breisgau, Albertstrasse 19, D-78 Freiburg im Breisgau, W. Germany) Thomas, C.; Sandritter, W. *Exp Pathol (Jena)* 13(2/3): 162-174; 1977.

Rhabdomyosarcomas induced in Wistar rats by sc or im injection of 10 mg of 9,10-dimethyl-1,2-benzanthracene were removed at 30, 60, 105, or 150 days postinjection and examined by light and electron microscopy. The chronological development of the tumors was characterized by four different cell types: myofibrillar cells, myofilamentous cells, undifferentiated sarcoma cells, and mature fibrosarcoma cells. The tumor cells contained dystrophic megamitochondria. Annulate lamellae typical of tumor cells were observed in the endoplasmic reticulum. Megacisternae in which protein-secreting material was condensed as a consequence of disturbed synthesis and secretion were also found. (33 refs.)

- 77-3155 **Increased In Vitro Growth Capacity of Tracheal Epithelium Exposed In Vivo to 7,12-Dimethylbenz(a)anthracene.** (Eng.) Marchok, A. C. (Biolog. Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830) Rhoton, J. C.; Griesemer, R. A.; Nettesheim, P. *Cancer Res* 37(6): 1811-1821; 1977.

Tracheal transplants from female Fischer 344 rats were implanted into sc pockets between the shoulders of isogenic recipient rats and, after several weeks, cylindrical beeswax pellets containing 7,12-dimethylbenz(a)anthracene (DMBA) were inserted into the lumens of the transplants. After 2 wk, explants were made from the exposed trachea and the in vitro growth characteristics were studied. During the initial planting, the rate of outgrowth was greatest from explants preexposed to 150  $\mu\text{g}$  DMBA. Outgrowth from explants preexposed to 640  $\mu\text{g}$  DMBA was sparse during the first planting, but it increased markedly during repeated planting when medium supplemented with insulin and hydrocortisone was used. Establishment of outgrowth during repeated planting was hormone-dependent, unlike that in explants derived from untreated control tracheas. Primary cultures could be established in 3/6 explants exposed to 150  $\mu\text{g}$  DMBA and 5/5 explants exposed to 640  $\mu\text{g}$  DMBA. Both primary cultures and subcultures obtained from the DMBA-treated tracheas exhibited the morphological characteristics of keratinizing squamous epithelium. Primary cultures obtained from control tracheas could not be subcultured. It is concluded that there is a marked increase in the in vitro growth capacity of tracheal epithelium after in vivo exposure to DMBA. (38 refs.)

- 77-3156 **Metabolism of 7,12-Dimethylbenz[a]anthracene (DMBA) by Rat Mammary Cells in Culture (Meeting Abstract).** (Eng.) Shepherd, R. E. (NCI Frederick Cancer Res. Center, Frederick, MD 21701) Bryan, A. H. *Proc Am Assoc Cancer Res* 18: 210; 1977. (no refs.)

- 77-3157 **Effect of Strain and Age on the Binding of 7,12-Dimethylbenz[a]anthracene (DMBA) to Rat Mammary Epithelial Cell Macromolecules (Meeting Abstract).** (Eng.) Janss, D. H. (NCI Frederick Cancer Res. Center, Frederick, MD 21701) Hadaway, E. I. *Proc Am Assoc Cancer Res* 18: 208; 1977. (no refs.)

- 77-3158 **The Effects of 7,8-Benzoflavone (7,8-BF) on Skin Tumor Initiating Activities of Various 7 and 12-Substituted Derivatives of 7,12-Dimethylbenz[a]anthracene (DMBA) (Meeting Abstract).** (Eng.) DiGiovanni, J. (Dept. Pharmacology, Univ. Washington, Seattle,

VA 98195) Viaje, A.; Juchau, M. R. *Proc Am Assoc Cancer Res* 18: 246; 1977. (no refs.)

7-3159 Tumor Development in Transplants of Rat Mammary Hyperplastic Alveolar Nodules (Meeting Abstract). (Eng.) Rivera, E. M. (Dept. Zoology, Michigan State Univ., East Lansing, MI 48824) Walbridge, M.; Hill, S. D. *Proc Am Assoc Cancer Res* 18: 203; 1977. (no refs.)

7-3160 8,9-Dihydro-DMBA-8,9-Diol: The Major Rat Liver Metabolite of DMBA (Meeting Abstract). (Eng.) Morreal, C. E. (Roswell Park Memorial Inst., Buffalo, NY 14263) Alks, V.; Dao, T. L. *Proc Am Assoc Cancer Res* 18: 241; 1977. (no refs.)

7-3161 Fluorescence of Isolated DMBA-DNA Products (Meeting Abstract). (Eng.) Moschel, R. C. (NCI, Frederick Cancer Res. Center, Frederick, MD 21701) Dipple, A. *Proc Am Assoc Cancer Res* 18: 177; 1977. (no refs.)

7-3162 Effect of Age of Non-skin Tissues on Susceptibility of Skin Grafts to 7,12-Dimethylbenz(a)anthracene (DMBA) Carcinogenesis in BALB/c Mice, and Effect of Age of Skin Graft on Susceptibility of Surrounding Recipient Skin to DMBA. (Eng.) Ibbesen, P. (Section Tumor Virus Res., Inst. Medical Microbiology, Univ. Copenhagen, 22 Juliane Maries vej, DK-100 Copenhagen, Denmark) *J Natl Cancer Inst* 58(4): 1057-1060; 1977.

The influence of age-dependent alterations in nonskin tissues on chemical carcinogen-induced skin papilloma development was studied by treating (DMBA, 32 µg in 25 microliter acetone) 4-mo-old skin grafts sewed onto 4- and 20-mo-old syngeneic recipients with 7,12-dimethylbenz(a)anthracene (DMBA). The skin of 4- and 20-mo-old BALB/c female mice differed in susceptibility to DMBA carcinogenesis, but the 4-mo-old grafts showed the same papilloma incidence independent of the age of the recipient mice. In a different experiment, the influence of skin graft age on papilloma development in DMBA-treated recipient skin was studied. Fourteen- and 26-mo-old skin grafts were carried by 14-mo-old recipients. Grafts of those two ages are known to differ in susceptibility to DMBA carcinogenesis, but no effect of the grafts on papilloma development in DMBA-treated recipients was detectable. It is concluded that certain age-dependent differences in skin susceptibility to chemical car-

cinogens are due to alterations in the skin at the site of carcinogen treatment. (15 refs.)

77-3163 Effects of 5-Fluorouracil (FU) on tRNA in a Transplantable Mouse Mammary Adenocarcinoma Induced by 7,12-Dimethylbenz(a)anthracene (DMBA) (Meeting Abstract). (Eng.) Tseng, W. -C. (Baylor Coll. Medicine, Houston, TX 77030) Medina, D.; Randerath, K. *Proc Am Assoc Cancer Res* 18: 105; 1977. (no refs.)

77-3164 Mammary Tumorigenesis in Chemical Carcinogen-treated Mice. VII. Prolactin and Progesterone Levels in BALB/c Mice. (Eng.) Medina, D. (Dept. Cell Biology, Baylor Coll. Medicine, Houston, TX 77030) O'Bryan, S. B.; Warner, M. R.; Sinha, Y. N.; VanderLaan, W. P.; McCormack, S.; Hahn, P. *J Natl Cancer Inst* 59(1): 213-219; 1977.

Polyacrylamide gel electrophoresis and specific radioimmunoassays were used to measure pituitary and serum prolactin and serum progesterone levels in BALB/c mice that had received 3-methylcholanthrene (MC) or dimethylbenz(a)anthracene (DMBA) intragastrically beginning at age 8 wk. Sera and pituitary glands were collected from female mice killed at age 15-17 wk or 44 wk. Neither 1.5 mg MC nor 1.5-6.0 mg DMBA significantly altered pituitary content or serum prolactin concentration in the old mice, but DMBA slightly increased (approx 33%) the total amount of pituitary prolactin in the 44-wk-old mice. The elevation of pituitary prolactin content in these mice was not correlated with the incidence of mammary tumors in the group or in an individual mouse. Serum progesterone levels were increased approx 22% in MC-treated mice by 50 days after the last treatment. This increase could be attributed to higher serum levels during diestrus and proestrus. Progesterone levels were unaltered by ovariectomy but were reduced approx 60% by adrenalectomy. DMBA had no significant effect on serum progesterone levels in mice assayed at 44 wk of age. These results gave little support to the concept that MC and DMBA promote murine mammary tumorigenesis by leading to a sustained increase in pituitary prolactin content or serum prolactin concentrations either shortly (15-17-wk-old mice) after carcinogen treatment or during mammary tumor formation and growth (44-wk-old mice). (38 refs.)

77-3165 Inhibition of Mammary Cancer by Retinyl Methyl Ether. (Eng.) Grubbs, C. J. (IIT Res. Inst., Chicago, IL 60616) Moon, R. C.; Sporn, M. B.; Newton, D. L. *Cancer Res* 37(2): 599-602; 1977.

The relative effectiveness of retinyl methyl ether and retinyl



acetate in preventing mammary cancer induced by 7,12-dimethylbenz(a)anthracene (DMBA) was studied in female Sprague-Dawley rats. Daily feeding of retinyl methyl ether [380 or 760 micromoles ( $\mu\text{mol}$ )/kg of diet] beginning 1 wk after the po administration of DMBA (5 and 15 mg), reduced the incidence of mammary cancer and the number of mammary tumors, both malignant and benign. Retinyl methyl ether also markedly increased the latent period for the appearance of mammary cancers. Retinyl methyl ether had no evident toxicity and did not affect wt gain. This synthetic retinoid was superior to the natural retinoid, retinyl acetate, for the inhibition of mammary carcinogenesis. At 380  $\mu\text{mol}$ /kg of diet, retinyl acetate reduced neither the incidence nor the total number of tumors in rats treated with 5 mg DMBA. (22 refs.)

- 77-3166 **Effects of Serum Concentration on the Expression of Carcinogen-induced Transformation in the C3H/10T1/2 CL8 Cell Line.** (Eng.) Bertram, J. S. (Dept. Experimental Therapeutics, Grace Cancer Drug Center, Roswell Park Memorial Inst., Buffalo, NY 14263) *Cancer Res* 37(2): 514-523; 1977.

The C3H/10T1/2 CL8 cell line (10T1/2), derived from mouse embryo fibroblasts, is widely used as a quantitative assay system for chemical and physical carcinogens. 10T1/2 cells, but not their transformed counterparts, exhibit a decreased final saturation density with decreasing serum concentration. Exposure of dimethylbenz(a)anthracene-treated cultures to 5% serum 8 days posttreatment led to a two- to sixfold enhancement in transformation frequency compared with cultures maintained in 10% serum throughout. Exposure 14, 21, or 28 days posttreatment also enhanced transformation frequency, provided a sufficient time was allowed for expression of the malignant phenotype. Exposure to 5% serum 1 or 2 days posttreatment did not lead to significant enhancement of transformation frequency. In contrast, exposure to 15% or 20% serum after 8 days virtually abolished the expression of malignancy; however, this inhibition could be reversed by 5% serum. Morphologically transformed foci isolated from cultures exposed to 5% serum produced clones in agarose with the same frequency as did foci isolated from cultures exposed to 10% serum. Reconstruction experiments using confluent monolayers of 10T1/2 cells overlaid with 3-methylcholanthrene transformed cells demonstrated that the growth of transformed cells decreased proportionally with the log of serum concentration. This effect was not caused by medium depletion, and it depended on the presence of 10T1/2 cells. It is concluded that the expression of malignancy in this system is governed by the serum-modulated cell density of the mass of nontransformed cells in the culture. (35 refs.)

- 77-3167 **Lung Cancer and the Aryl Hydrocarbon Hydroxylase Polymorphism (Meeting Abstract).**

(Eng.) Paigen, B. (Roswell Park Memorial Inst., Buffalo, NY 14263) Gurtoo, H. L.; Minowada, J.; Vincent, R.; Paigen, K.; Houten, L. *Proc Am Assoc Cancer Res* 18: 206; 1977. (no refs.)

- 77-3168 **Distribution of Aryl Hydrocarbon Hydroxylase Inducibility in Cultured Human Lymphocytes.**

(Eng.) Paigen, B. (Dept. Molecular Biology, Roswell Park Memorial Inst., Buffalo, NY 14263) Minowada, J.; Gurtoo, H. L.; Paigen, K.; Parker, N. B.; Ward, E.; Hayner, N. T.; Bross, I. D.; Bock, F.; Vincent, R. *Cancer Res* 37(6): 1829-1837; 1977.

Aryl hydrocarbon hydroxylase (AHH) activities were measured in untreated lymphocytes and lymphocytes treated with the AHH-inducer, 3-methylcholanthrene, from 53 human donors. Considerable seasonal variation was seen in both basal and induced AHH levels and in AHH inducibility (induced level/basal level) when either values for lymphocytes from the same donors were considered or values for the total donor population were considered. Thus, induced AHH levels from January to May inclusive represented only about 20% of the values obtained throughout the rest of the year. When measurements were limited to the summer and fall seasons, when AHH activities are high, AHH inducibility was found to be reproducible for most people studied, with repeat determinations for the same person falling within 11% of the mean value. Values of AHH inducibility ranged from 0.9 to 5, but their distribution did not fall into three distinct classes, as had been reported. AHH inducibilities could not be assessed in lung cancer patients, since lymphocytes from these patients did not respond well to mitogen stimulation in vitro. (29 refs.)

- 77-3169 **3-Methylcholanthrene-induced Monooxygenase (O-Deethylation) Activity of Human Lymphocytes.** (Eng.) Burke, M. D. (Forensic Science Dept., Karolinska Institutet, S-10401 Stockholm, Sweden) Mayer, R. T.; Kouri, R. E. *Cancer Res* 37(2): 460-463; 1977.

Mixed-function oxidase (MFO) activity was determined in mitogen-activated human lymphocytes by a direct fluorescence assay. The O-deethylation of ethoxyresorufin to resorufin was used to quantitate MFO activity. Ethoxyresorufin O-deethylase activity was low to nondetectable in noninduced, mitogen-activated cells, but it was readily detected in 3-methylcholanthrene-treated, mitogen-activated lymphocytes. The activity was (1) dependent on assay time and number of lymphocytes; (2) dependent on the presence of NADPH; (3) stable to freezing at -80 C for at least 2 wk; (4) reproducibly detected in duplicate samples of blood from one individual when cultured and assayed at the same time; but (5) quite variable in samples of blood from one individual at different times. Since ethoxyresorufin is a specific substrate for cytochrome P-448-associated monooxygenase in hepatic and pul-

monary tissue of model animal systems, the use of this chemical could proffer an assay that specifically measures human cytochrome P-448-associated activity. (26 refs.)

**77-3170 Metabolism of [<sup>3</sup>H]-3-Methylcholanthrene in the Perfused Rat Liver.** (Eng.) Takahashi, G. (Dept. Pathology, Chest Disease Res. Inst., Kyoto Univ., Kyoto, Japan) Shah, H.; Weinhouse, S. *Cancer Res* 37(2): 369-375; 1977.

The metabolism of <sup>3</sup>H-3-methylcholanthrene (3-MC) was studied in isolated, perfused Charles River rat livers. Following addition of 250 µg to the perfusion fluid, 3-MC disappeared rapidly. After 2 hr, approx 34% of the radioactivity was excreted in the bile, 6% remained in the perfusate, and 60% was found in the liver. Of the liver radioactivity, 80% was unchanged 3-MC, 11% was conjugated metabolites, 4% was free hydroxymetabolites, and 4% was nonextractable, presumably bound to macromolecules. Of the perfusate radioactivity, 82% was conjugated metabolites, 3% was free hydroxymetabolites, and 15% was unchanged 3-MC. A similar distribution was observed in intact, bile-cannulated rats, but biliary excretion was about one-fourth as high with double the iv-injected dose. Biliary excretion in perfused livers rose rapidly during the first 30-40 min, then decreased steadily. It was nearly twice as high in male as in female rat livers. Pretreatment of rats with 3-MC more than doubled the biliary excretion rate in livers of both sexes over the first 20-30 min and raised that of the female to that of the male rat liver. Neither retinol acetate nor 7,8-benzoflavone had any appreciable effect on the biliary excretion of 3-MC metabolites. 2-Diethylaminoethyl 2,2-diphenylvalerate, a well-known microsomal oxygenase inhibitor, lowered excretion by 80%-90% and lengthened the lag period: dibutyl cyclic AMP markedly increased the rate of excretion of 3-MC metabolites. Fractionation of the bile by chromatography on Sephadex LH-20 revealed six well-defined peaks of radioactivity. In contrast, bile of intact rats given 3-MC gave a pattern consisting of only three peaks. Preliminary data suggest that these consist of conjugates of dihydroxymetabolites as well as more highly hydroxylated derivatives. The data indicate that the perfused liver is an appropriate experimental model for studies on the hepatobiliary metabolism of carcinogens. (29 refs.)

**77-3171 The Uptake and Secretion of 3-Methylcholanthrene by the Prostate Glands of the Rat and Dog.** (Eng.) Smith, E. R. (Dept. Pharmacology, Univ. Massachusetts Medical Sch., 55 Lake Ave. N., Worcester, MA 01605) Hagopian, M. *J Natl Cancer Inst* 59(1): 119-122; 1977.

Prostatic fluid was collected in four anesthetized Sprague-Dawley rats over a 2-hr interval from 24 to 26 hr after a single

ip dose of [6-<sup>14</sup>C]-3-methylcholanthrene (<sup>14</sup>C-MC, 5 mg/kg). During this time, the radioactivity levels in the fluid were only slightly less than those in plasma; at 26 hr after treatment, levels of radioactivity within the prostate were higher than those in the prostatic fluid or plasma. When unanesthetized dogs with surgically prepared prostatic fistulas were given a single ip dose of 0.5 mg/kg <sup>14</sup>C-MC, and when serial prostatic fluid and plasma samples were collected over the ensuing 50 hr (2 dogs) or 212 hr (1 dog), the radioactivity levels in the prostatic fluid were initially greater than those in plasma, but then they fell progressively with time to less than those of plasma. At 50 hr after treatment, radioactivity was recovered from the prostate glands of two dogs with a fistula and two dogs without a fistula at levels of about one-fourth those of plasma. Thus, <sup>14</sup>C-MC and/or its metabolites enters the prostate glands and prostatic fluids of the rat and dog. (13 refs.)

**77-3172 Immunologic Status, Age and 3-MC Tumorigenesis in NZB Mice (Meeting Abstract).** (Eng.) Morton, J. I. (Univ. Oregon Health Sciences Center, Portland, OR 97201) Siegel, B. V.; Moore, R. D. *Proc Am Assoc Cancer Res* 18: 210; 1977. (no refs.)

**77-3173 Reversible Expression of Transformation in Cells Grown in a Chemically Defined Medium (Meeting Abstract).** (Eng.) Tomei, L. D. (Grace Cancer Drug Center, Roswell Park Memorial Inst., Buffalo, NY 14263) Bertram, J. S. *Proc Am Assoc Cancer Res* 18: 244; 1977. (no refs.)

**77-3174 Induction of Epithelial Neoplasms in Hamster Tracheal Grafts with 3-Methylcholanthrene (MC) Coated Lycra Fibers (Meeting Abstract).** (Eng.) Mossman, B. T. (Univ. Vermont, Burlington, VT 05401) Craighead, J. E. *Proc Am Assoc Cancer Res* 18: 222; 1977. (no refs.)

**77-3175 Preparation and Characterization of Total, Rough and Smooth Microsomes from the Lung of Control and Methylcholanthrene-treated Rats.** (Eng.) Johansson, K. (Arrhenius Lab., Dept. Biochemistry, Univ. Stockholm, Fack, 104 05 Stockholm, Sweden) DePierre, J. W.; Bergstrand, A.; Dallner, G.; Ernster, L. *Biochim Biophys Acta* 496(1): 115-135; 1977.

Optima conditions for the preparation of relatively pure microsomes and microsomal subfractions from the rat lung



were determined. The most important condition is homogenization of a 20% wt/volume (w/v) suspension of lung tissue in 0.44 M sucrose/1% (w/v) bovine serum albumin with four up-and-down strokes at 440 revolutions/min in a Potter-Elvehjem homogenizer. The 10,000 x g supernatant prepared from this homogenate can be centrifuged at 105,000 x g to obtain total microsomes or subfractionated into rough and smooth microsomes on a Cs<sup>+</sup>-containing discontinuous sucrose gradient. The total, rough, and smooth microsomes from Sprague-Dawley rat liver were characterized in terms of their chemical composition, enzymatic activity, and morphology. Methylcholanthrene (20/mg/kg, ip) affected neither the quantity of endoplasmic reticulum membrane nor the level of NADPH-cytochrome c reductase in rat lung. The polycyclic hydrocarbon appeared to induce benzpyrene monooxygenase activity to a greater extent in the smooth endoplasmic reticulum than in the rough endoplasmic reticulum. The microsome preparations should be useful in studies of the distribution of benzpyrene monooxygenase, epoxide hydase, and enzymes of phospholipid and ascorbic acid synthesis. (48 refs.)

- 77-3176 **Transformation and Neoplastic Development of Hamster Chondrocytes after Exposure to 4-Nitroquinoline-1-oxide and 3-Methylcholanthrene in Tissue Culture.** (Eng.) Katoh, Y. (Dept. Experimental Pathology, Cancer Inst., 1-37-1 Kami-Ikebukuro, Toshima-ku, Tokyo 170, Japan) *J Natl Cancer Inst* 59(1): 155-163; 1977.

Sternal hyaline cartilages of Syrian hamsters were dissociated with collagenase and cultured. Primary monolayer cultures of the dissociated cells were morphologically homogeneous. Secondary cultures of the chondrocytes were treated with  $1 \times 10^{-6}$  or  $2 \times 10^{-6}$  M 4-nitroquinoline-1-oxide (4NQO) for 3 hr or with 5 or 10  $\mu$ g 3-methylcholanthrene (MC)/ml for 3 days. Cultured chondrocytes were transformed morphologically 29-51 days after 4NQO treatment and 41-61 days after MCA treatment. The transformed cells began to grow continuously in vitro, and they had a fibroblastic appearance. Untreated cells and cells treated with dimethyl sulfoxide did not transform within at least 110 days after inoculation. Among the transformed cells, one near-diploid cell line preserved the distinct phenotypic expression of chondrocytes, but heteroploid cell lines lost their differentiated features. The near-diploid cell line produced nodules in the cheek pouches of hamsters within 1 wk, but the nodules later regressed. They showed the chondrogenic properties of the original cells and stained metachromatically with toluidine blue. Heteroploid cell lines formed progressive tumors with few chondrogenic features; these tumors were diagnosed as fibrosarcomas. (39 refs.)

- 77-3177 **On the Significance of Sex Hormones in Producing Experimental Prostate Tumor in the**

**Rat.** (Eng.) Higuchi, M. (Dept. Urology, Kurume Univ. Medical Sch., Kurume, Japan) *Recent Results Cancer Res* 60: 27-52; 1977.

The influence of sex hormones on experimental prostate tumor formation was investigated in Donryu rats, and the effects of changes in endocrine milieu on transplantation of experimentally formed prostate adenoma were examined. In rats receiving direct injection of carcinogens to the ventral lobe of the prostate, tumors occurred in 50% of the animals with 20-methylcholanthrene (20-MC) and 51.3% with 4-nitroquinoline-N-oxide (4-NQO). In rats receiving transplanted wrapped carcinogens, tumor formation was 27.8% with 20-MC and 19.6% with 4-NQO. There was no significant difference in tumor formation between rats receiving carcinogens only and those undergoing androgen treatment, castration, or estrogen treatment after castration. The tumors were histologically classified as adenomas, squamous cell carcinomas, and sarcomas. Prostatic adenocarcinoma, which constitutes most human prostate cancers, was not induced by this treatment. In rats given hexestrol after castration, the successful transplantation rate was 60% and the rate of change from adenoma to adenocarcinoma was 20%. In female rats, the successful transplantation rate was 40% and the rate of adenocarcinoma, 4%. Estrogen dominance promoted the transition from adenoma to adenocarcinoma. It could not be determined whether the adenoma used was in a precancerous state, and the carcinogenic progress from adenoma to adenocarcinoma was not confirmed. (35 refs.)

- 77-3178 **Mutagenicities of Quinoline and Its Derivatives.** (Eng.) Nagao, M. (Biochemistry Div., Natl. Cancer Center Res. Inst., Tsukiji 5-1-1, Chuo-ku, Tokyo 104, Japan) Yahagi, T.; Seino, Y.; Sugimura, T.; Ito, N. *Mutat Res* 42(3): 325-342; 1977.

Quinoline, recently reported to be carcinogenic in rats, was mutagenic to *Salmonella typhimurium* tester strains TA100 and TA98 in the presence of a metabolic activation system, S-9 mix, prepared from liver homogenates of Sprague-Dawley rats inoculated ip with Kanechlor 500. 2-Chloroquinoline, a noncarcinogen, was nonmutagenic with or without S-9 mix. 8-Hydroxyquinoline, which is not known to be carcinogenic, was mutagenic with S-9 mix to both bacterial strains. The mutagenicities of 17 other quinoline derivatives that are not known to be carcinogenic were tested, and 12 were found to be mutagenic. (28 refs.)

- 77-3179 **In Vitro Production of Nucleolar Caps in the Trophoblast Cells of Early Mouse Embryo By 4-Nitroquinoline 1-Oxide (Meeting Abstract).** (Eng.) Chatterjee, A. (New York Univ., New York, NY 10003) *Diss Abstr Int [B]* 37(9): 4273-4274; 1977. (no refs.)

77-3180 **Excision Repair of DNA Base Damage in Human Cells Treated with the Chemical Carcinogen 4-Nitroquinoline 1-Oxide.** (Eng.) Ikenaga, M. (Dept. Fundamental Radiology, Faculty Medicine, Osaka Univ., Kita-ku, Osaka 530, Japan) Takebe, H.; Ishii, Y. *Mutat Res* 43(3): 415-427; 1977.

An investigation was made to determine whether DNA damage induced by the carcinogen 4-nitroquinoline 1-oxide (4NQO) is repaired in human cells by an excision-repair system similar to the one in *Escherichia coli*. The initial formation and subsequent disappearance of 4NQO-nucleoside adducts were investigated in normal human amnion FL cells and in xeroderma pigmentosum (XP) cells unable to repair UV-induced pyrimidine dimers. Initially, two peaks of stable 4NQO-guanine adducts, one peak of a stable 4NQO-adenine adduct, and a peak due to 4-aminoquinoline 1-oxide (4AQO) released from a labile 4NQO-guanine fraction were seen on chromatograms of hydrolyzed DNA from both cell lines. The three kinds of stable 4NQO-purine adducts disappeared from the DNA of the normal FL cells at almost the same rate (approx 60% loss after 24 hr); the 4AQO adduct disappeared more rapidly. In the XP cells, however, the stable adducts did not disappear at all from the DNA, but about 40% of the 4AQO adduct did disappear in 24 hr. It is concluded that 4NQO-purine adducts are excised from normal human cells by the same excision-repair system that works on pyrimidine dimers. (30 refs.)

77-3181 **Inhibitory Effect of Caffeine on Chemical Carcinogenesis in Mice (Meeting Abstract).** (Eng.) Nomura, T. (Osaka Univ., Osaka 553, Japan and Univ. Wisconsin, Madison, WI 53706) *Proc Am Assoc Cancer Res* 18: 244; 1977. (no refs.)

77-3182 **Chemical Studies on Tobacco Smoke. XLII. Nitrosonornicotine: Presence in Tobacco, Formation and Carcinogenicity.** (Eng.) Hoffmann, D.; Hecht, S. S.; Ornaf, R. M.; Wynder, E. L.; Tso, T. C. In: *Environmental N-Nitroso Compounds: Analysis and Formation. Proceedings of a Working Conference held at the Polytechnical Institute, Tallinn, Estonian SSR, 1-3 October 1975*. Walker, E. A.; Bogovski, P.; Gričiute, L.; Davis, W., eds. International Agency for Research on Cancer. (Lyon, France) IARC Scientific Publication No. 14, pp. 307-320; 1976.

The presence and analysis of nonvolatile N-nitroso compounds in tobacco, the formation of nitrosonornicotine (NNN) in tobacco, and data on the carcinogenicity of the nitrosamines of nornicotine and anabasine are discussed. NNN was found in smoking tobaccos, chewing tobaccos, and snuff in concentrations of 0.3-90 µg/g. Data from a variety of sources suggest that NNN and other nitrosamines are

formed during curing and that nitrate content is an important factor in nitrosamine formation. Lung adenomas were seen in 28/35 NNN-treated (total dose 22 mg) female A/He mice, 10/32 control mice, and in 13/31 and 9/25 mice treated with 22 mg of N'-carbomethoxynornicotine and 2.2 mg of nornicotine, respectively. NNN was also carcinogenic to the esophagus and nasal cavity of rats. The presence of NNN in tobacco and its carcinogenicity in rodents are consistent with the possibility that NNN may be a significant factor in the increased risk of tobacco chewers to cancer of the esophagus. (24 refs.)

77-3183 **Investigations of Cigarette Smoke Dosages in Inhalation Experiments with Syrian Hamsters. II. Uptake of Cigarette Smoke by Animals; Comparisons on the Influence of Gas Phase and the Whole Smoke of Different Cigarettes.** (Eng.) Klimisch, H. J. (BASF Aktiengesellschaft-WOT-J 560, D-6700 Ludwigshafen-Rhein, W. Germany) Döntenwill, W. *J Natl Cancer Inst* 58(4): 935-939; 1977.

The increased CO<sub>2</sub> concentration (CO<sub>2</sub> enrichment) of the cigarette smoke-air mixture in an inhalation chamber containing Syrian hamsters was measured. The additional CO<sub>2</sub> originated in the animals' expiratory air and was considered a function of respiratory capacity. This method of characterizing the respiratory behavior of animals in inhalation chambers indicated that the vapor phase of cigarette smoke reduces respiratory capacity by up to 45%, whole smoke by up to 60%. Comparative investigations with different cigarettes showed that qualitative and quantitative differences in their gas and vapor phases did not induce measurable differences in respiratory behavior. Thus, inhalation experiments are suitable for comparing the relative effects of different cigarette types. Estimates of the amount of total particulate matter inhaled were similar to those obtained in experiments with radioactively labeled smoke. (10 refs.)

77-3184 **Dehydroretronecine Induced Skin Tumors in Mice (Meeting Abstract).** (Eng.) Robertson, K. A. (Univ. Wisconsin, Madison, WI 53706) Johnson, W. D. *Proc Am Assoc Cancer Res* 18: 213; 1977. (no refs.)

77-3185 **Malignant Neoplasms in Rats Fed Lasiocarpine (Meeting Abstract).** (Eng.) Rao, M. S. (Northwestern Univ. Medical Sch., Chicago, IL) Reddy, J. K. *Am J Pathol* 86(2): 24a-25a; 1977. (1 ref.)



- 77-3186 Research on the Carcinogenic Activities of N-Nitrosopyrrolidine, N-Nitroso-2-pyrrolidone, and N-Nitroso-5-methyl-2-pyrrolidone. (Jpn.) Takatori, K. (Dept. Pharmacy, Nagoya Univ. Hosp., Tsurumai-cho, Showa-Ku, Nagoya, Japan) Mori, H.; Kato, T.; Hasegawa, T.; Nakano, S.; Hirono, I. *J Pharm Soc Jpn* 97(3): 320-323; 1977.

N-Nitrosopyrrolidine (I), N-nitroso-2-pyrrolidone (II), and N-nitroso-5-methyl-2-pyrrolidone (III) were administered in drinking water to male Sprague-Dawley rats for 365 days. In animals surviving > 276 days, I induced hepatocellular carcinoma in 14/18 animals and II induced colon carcinoma in 1/15 animals, but III did not induce carcinoma in any of 16 animals. The LD50 (po) increased in the order II (291 mg/kg), III (405 mg/kg), and I (1,039 mg/kg), and the mutagenic activity (recombinant assay in *Bacillus subtilis* mutants H-17 and M-45) increased in the order I, III, and II. These orders have little relation to the carcinogenicity of the three compounds. (15 refs.)

- 77-3187 Mutagenesis by Nitrosocarbaryl and Related Compounds in *Escherichia Coli* and *Haemophilus Influenzae* (Meeting Abstract). (Eng.) Elespuru, R. K. (Univ. Tennessee, TN) *Diss Abstr Int [B]* 37(8): 3767; 1977. (no refs.)

- 77-3188 Evidence for the Formation of Mutagenic N-Nitroso Compounds in the Human Body (Meeting Abstract). (Eng.) Varghese, A. J. (Ontario Cancer Inst., Toronto 5, Ontario, Canada) Land, P.; Furrer, R.; Bruce, W. R. *Proc Am Assoc Cancer Res* 18: 80; 1977. (no refs.)

- 77-3189 Nitrite and Thiocyanate in the Fasting and Secreting Stomach and in Saliva. (Eng.) Ruddell, W. S. (Dept. Gastroenterology, Central Middlesex Hospital, London, England) Blendis, L. M.; Walters, C. L. *Gut* 18(1): 73-77; 1977.

N-nitroso compounds are carcinogenic in most animal species tested. Nitrite and thiocyanate levels influence the rate of nitrosation. The nitrite and hydrogen ion concentrations in the fasting gastric juice of 17 consecutive patients undergoing routine pentagastrin tests were measured. Ten patients had chronic duodenal ulcers, three had chronic gastric ulcers, including one with additional duodenal ulcer, and in five patients no evidence of gastroduodenal ulcer was found. No patient had gastric cancer. Of these 17 patients 12 were smokers. Nitrite was found in all of 17 samples of fasting gastric juice; mean  $4.9 \pm 1.1 \mu\text{M}$ . Stimulation of gastric secretion

with pentagastrin caused no significant change in nitrite concentration. Thiocyanate was detected in all of 21 samples of fasting gastric juice. The difference in concentration ( $p < 0.02$ ) between smokers ( $1.1 \pm .01 \text{ mM}$ ) and non-smokers ( $0.4 \pm .01 \text{ mM}$ ) probably reflects similar differences in saliva. In contrast to the nitrite data there was a significant drop in the mean thiocyanate concentration of gastric juice after pentagastrin, from  $0.9 \pm .04 \text{ mM}$  to  $0.3 \pm .04 \text{ mM}$ , suggesting a salivary origin for the thiocyanate in gastric juice. Thiocyanate at 1 mM is a powerful catalyst of nitrosation, which, together with small amounts of nitrite and naturally occurring amines, could lead to the intragastric formation of carcinogenic nitrosamines and in certain circumstances be a factor in the etiology of gastric cancer. (22 refs.)

- 77-3190 The Effects of Additional Flora on the Response of *Salmonella* Mutants Lodged in the Gastrointestinal Tract. (Eng.) Wheeler, L. A. (Dept. Pharmacology, Beth Israel Hosp., 330 Brookline Ave., Boston, MA 02215) Carter, J. H.; Ingelfinger, J. A.; Soderberg, F. B.; Goldman, P. *Cancer Res* 37(2): 451-455; 1977.

A histidine auxotroph of *Salmonella typhimurium*, strain TA1538, will lodge for several months in the gastrointestinal tract of otherwise germ-free rats and of rats additionally associated with bacteria characteristic of normal flora such as *Lactobacillus plantarum* and *Bacteroides vulgatus*. In the presence of the additional flora, the concentration of strain TA1538 is diminished in the stomach but not in the lower gastrointestinal tract or feces. Experiments showed that following the ingestion of 2-nitrofluorene ( $3.4$  or  $34 \text{ mg}$ ), there was an increase in the concentration of revertants in the feces that reflected that observed in the colon and cecum. A dose-response relationship was demonstrated between the amount of 2-nitrofluorene ingested and the concentration of revertants in the feces. A given dose of 2-nitrofluorene, however, produced fewer revertants in the feces of rats with the additional flora than in the feces of rats associated only with strain TA1538. It is not clear whether the decreased number of revertants in the feces in the presence of the additional flora is a result of metabolic transformations of 2-nitrofluorene by *B. vulgatus*, which can be demonstrated in vitro, or a result of displacement of strain TA1538 from the stomach. Rats associated with strain TA1538 or other Ames tester strains may be useful for detecting carcinogens as mutagens within the gastrointestinal tract and for determining the influence of various constituents of bacterial flora on the concentration of mutagenic compounds. (15 refs.)

- 77-3191 Free Radicals and Carcinogenesis: Nitroxyl Free Radical Formation in a Nitrosofluorene-unsaturated Lipid Reaction (Meeting Abstract). (Eng.)

Floyd, R. A. (Biomembrane Res. Lab., Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104) *Biophys J* 17(2): 168; 1977. (no refs.)

**77-3192 Kinetics and Mechanism of Carbon-8 Methylation of Purine Bases and Nucleosides by Methyl Radical.** (Eng.) Zady, M. F. (Dept. Chemistry, Univ. Louisville, Louisville, KY 40208) Wong, J. L. *J Am Chem Soc* 99(15): 5096-5101; 1977.

The mechanism and kinetics of the free-radical methylation of caffeine, adenine, guanine, hypoxanthine, adenosine, guanosine and inosine are presented. The findings provide a quantitative model for the molecular investigations of chemical carcinogenesis. (23 refs.)

**77-3193 Occupationally Induced Lung Carcinoma Following Inhalation of Alkylating Compounds (Meeting Abstract).** (Ger.) Bettendorf, U. (Wiesbaden, W. Germany) *Zentralbl Allg Pathol* 121(3): 298-299; 1977. (no refs.)

**77-3194 Formation of Nitrosamines by Interaction of Some Drugs with Nitrite in Human Gastric Juice.** (Eng.) Scheunig, G.; Ziebarth, D. In: *Environmental N-Nitroso Compounds: Analysis and Formation. Proceedings of a Working Conference held at the Polytechnical Institute, Tallinn, Estonian SSR, 1-3 October 1975.* Walker, E. A.; Bogovski, P.; Gričiute, L.; Davis, W., eds. International Agency for Research on Cancer. (Lyon, France) IARC Scientific Publication No. 14, pp. 269-277; 1976.

To determine the possible extent of nitrosation in the human stomach, which can lead to the formation of putative carcinogens, various drugs were reacted with nitrite in human gastric juice in vitro. The drugs were incubated with 1.6 micromoles of nitrite in 100 ml of gastric juice for 1 hr at 37 C and pH 2. The extents of conversion to nitrosamine were 69.0% for aminopyrine, 11.0% for analgin, and 74.8% for piperazine. In addition to the main product, dimethylnitrosamine, obtained with aminopyrine, a second weakly volatile unidentified N-nitroso compound, was demonstrated by thin layer chromatography. No nitrosation occurred with oxytetracycline or phenmetrazine hydrochloride. The pH-dependence and nitrite-consumption rate of the reactions were also studied. In conclusion, it is suggested that drugs containing secondary or tertiary amines should not be ingested unless absolutely necessary. (18 refs.)

**77-3195 Metabolism of Aliphatic and Cyclic Nitrosamines in Cultured Human Bronchus (Meeting Abstract).** (Eng.) Harris, C. C. (Human Tissue Studies Section, Experimental Pathology Branch, NCI, Bethesda, MD 20014) Autrup, H.; Stoner, G. *Proc Am Assoc Cancer Res* 18: 85; 1977. (no refs.)

**77-3196 Rapid Analysis of Methylated Purines and N-Methylnicotinamide in Rat Urine Following In Vivo Administration of Methylating Agents (Meeting Abstract).** (Eng.) Shaikh, B. (NCI, Frederick Cancer Res. Center, Frederick, MD 21701) Zielinski, W. L. *Proc Am Assoc Cancer Res* 18: 93; 1977. (no refs.)

**77-3197 Effects of Inhibitors on HCHO Formation and Macromolecular Binding During Oxidative Demethylation of Dimethylnitrosamine In Vitro (Meeting Abstract).** (Eng.) Lotlikar, P. D. (Fels Res. Inst., and Dept. Biochemistry, Temple Univ. Sch. Medicine, Philadelphia, PA 19140) Baldy, W. J.; Hong, Y. S.; Kim, S. *Proc Am Assoc Cancer Res* 18: 217; 1977. (no refs.)

**77-3198 Length of In Vivo Exposure to a Carcinogenic Dose of Dimethylnitrosamine Necessary for Subsequent Expression of Morphological Transformation by Rat Kidney Cells In Vitro.** (Eng.) Hard, G. C. (Dept. Pathology, Univ. Melbourne Medical Centre, Grattan St., Parkville, 3052, Victoria, Australia.) King, H.; Borland, R.; Stewart, B. W.; Dobrostanski, B. *Oncology* 34(1): 16-19; 1977.

An investigation was made of in vitro growth properties relevant to transformation in rat kidney cells isolated at intervals from 1 to 24 hr after a single carcinogenic dose of dimethylnitrosamine (DMN: 60 mg/kg ip to Porton albino Wistar rats). Morphological transformation, as indicated by development of large, dense foci of rapidly proliferating multilayered cells, occurred in all lines. In cultures isolated 4 hr or more after DMN administration, morphological transformation appeared consistently at subculture 5, but its expression was delayed to subculture 6 in the 2- and 3-hr cultures and to subculture 7 in the 1-hr cultures. Relative to control kidney cell cultures, transformed cells exhibited enhanced proliferative properties that were evident from assays of plating efficiency and DNA synthesis. The capacity for increased cloning efficiency by the test cultures was acquired several subcultures after the expression of morphological transformation, and colony-formation ability in semisolid media evolved later still. The results suggest that target cells perma-



nently altered by the carcinogen are present as early as 1 hr after the administration of DMN, but that the effect is more significant by 4 hr. (20 refs.)

- 77-3199 **Persistence of Carcinogen Induced Lesions in the DNA of Target Organ (Meeting Abstract).** (Eng.) Abanobi, S. E. (Fels Res. Inst., Dept. Biochemistry, Temple Univ. Medical Sch., Philadelphia, PA 19140) *Proc Am Assoc Cancer Res* 18: 248; 1977. (no refs.)
- 77-3200 **Virus-Like Particle (C Type) in Dimethylnitrosamine-Treated Organ Culture of Embryonic Rat Lung (Meeting Abstract).** (Eng.) Parsa, I. (State Univ. New York Coll. Medicine, Downstate Medical Center, Brooklyn, NY) *Am J Pathol* 86(2): 66a-67a; 1977. (no refs.)
- 77-3201 **Studies on the Induction of Chromosomal Aberrations and Sister Chromatid Exchanges by Indirectly Acting Carcinogens in Chinese Hamster Cells in the Presence of Rat-Liver Microsomes (Meeting Abstract).** (Eng.) Natarajan, A. T. (Dept. Radiation Genetics and Chemical Mutagenesis, State Univ. Leiden, Netherlands) *Mutat Res* 46(2): 144; 1977. (no refs.)
- 77-3202 **In Vivo Carcinogen-Induced Rat Liver Changes Studied In Vitro (Meeting Abstract).** (Eng.) Borenfreund, E. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021) *In Vitro* 13(3): 181-182; 1977. (no refs.)
- 77-3203 **Mitotic Rates in Infant Mouse Livers Following Oral Administration of Diethylnitrosamine or Its Precursors (Meeting Abstract).** (Eng.) Rijhsinghani, K. S. (Dept. Pathology, Michael Reese Medical Center, Chicago, IL 60616) Jao, W.; Swerdlow, M. A.; Rao, K. V.; Veselinovitch, S. D. *Proc Am Assoc Cancer Res* 18: 217; 1977. (no refs.)
- 77-3204 **Experimental Carcinoma of Liver in Monkeys (Meeting Abstract).** (Eng.) Gyorkey, F. (Veterans Admin. Hosp., Houston, TX) Mirkovic, R.; Uribe, G.; Min, K. W.; Gyorkey, P.; Melnick, J. L. *Am J Pathol* 86(2): 68a-69a; 1977. (no refs.)
- 77-3205 **Comparison of the Blood Supply to Diethylnitrosamine-induced Hyperplastic Nodules and Hepatomas and to the Surrounding Liver.** (Eng.) Solt, D. B. (Dept. Pathology, Univ. Toronto, Toronto, Ontario, Canada M5S 1A8) Hay, J. B.; Farber, E. *Cancer Res* 37(6): 1686-1691; 1977.
- The blood supply to diethylnitrosamine (DEN)-induced hyperplastic liver nodules and hepatomas was compared with the blood supply to the surrounding normal liver tissue of Fischer 344 rats. Radiolabeled microspheres of Sephadex were injected into the heart or portal veins to provide a quantitative index of arterial and portal blood supplies. The portal blood supply to 25 selected DEN-induced lesions was only 39% of that to the surrounding normal liver tissue; there was no apparent relationship between blood supply and lesion size or histological appearance. The arterial blood supply to lesions was similar to that to surrounding normal tissue. The fraction of the cardiac output received by lung, kidneys, spleen, and liver was similar in both control and DEN-treated rats. Although only 0.13% of the microspheres injected via the portal system were recovered in the lungs of control rats, about 100 times this number lodged in the lungs of rats with livers containing nodules and hepatomas. Alterations in the pattern of blood flow through neoplastic livers could contribute to biological diversification of hepatic lesions in successive stages of cancer evolution and could facilitate metastasis from the liver. (17 refs.)
- 77-3206 **Morphology of Diethylnitrosamine-induced Lung Tumours in Dzungarian Dwarf Hamsters.** (Eng.) Warzok, R. (Medical Acad. Erfurt, Inst. Pathology, Res. Group Preventive Oncology, Nordhauser Strasse 74, DDR-50 Erfurt, E. Germany) Thust, R. *Exp Pathol (Jena)* 18(1): 44-51; 1977.
- After transplacental application of 30 mg/kg diethylnitrosamine, 10/21 Dzungarian dwarf hamsters developed lung tumors. The tumors were classified as papillary, tubular, or alveolar adenomas or adenocarcinomas. Different histologic structures occurred in the same animal and even in different areas of the same tumors. The neoplasms derived from epithelial outgrowths of the small bronchioles. The significance of the Dzungarian hamster as a suitable model for transplacental and postnatal cancer research is discussed. (26 refs.)
- 77-3207 **Experimental Pancreatic Carcinogenesis. I. Morphogenesis of Pancreatic Adenocarcinoma**

in the Syrian Golden Hamster Induced by N-Nitroso-bis(2-hydroxypropyl)amine. (Eng.) Levitt, M. H. (Building 37, Room 3A19, NCI, NIH, Bethesda, MD 20014) Harris, C. C.; Squire, R.; Springer, S.; Wenk, M.; Thomas, D.; Kingsbury, E.; Newkirk, C. *Am J Pathol* 88(1): 5-28; 1977.

In a serial sacrifice experiment, outbred male Syrian golden hamsters were treated for life with weekly sc injections of N-Nitroso-bis(2-hydroxypropyl)amine (DIPN: 250 mg/kg in olive oil or deionized water). The pancreas was examined by high-resolution light (1-micron sections) and transmission electron microscopy. Early nonspecific changes in all pancreatic epithelial cellular elements were followed by a progressive proliferation of intra- and interlobular duct cells, with the development of multicentric foci of cystic and papillary cystic adenomas, intraductal carcinomas, and invasive ductal neoplasms. The progression to overt neoplasms was accelerated when DIPN was administered in H<sub>2</sub>O (10 wk, vs 22 wk for DIPN in olive oil). These observations are consistent with a multistage morphogenesis of pancreatic adenocarcinoma of ductal origin. (12 refs.)

77-3208 Angiosarcoma of the Liver in Guinea Pigs Induced by 2,2'-Dihydroxy-Di-N-Propylnitrosamine-Light and Electron Microscopic Features (Meeting Abstract). (Eng.) Rao, M. S. (Dept. Pathology, Northwestern Univ. Medical Sch., Chicago, IL 60611) Reddy, J. K. *Fed Proc* 36(3): 348; 1977. (no refs.)

77-3209 The Mutagenicity of Methylbenzyl Nitrosamine and Its  $\alpha$ -Acetoxy Derivatives. (Eng.) Tannenbaum, S. R. (Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA 02139) Kraft, P.; Baldwin, J.; Branz, S. *Cancer Lett (Amsterdam)* 2(6): 305-310; 1977.

The biological activity of the  $\alpha$ -acetoxy derivatives of methylbenzyl nitrosamine was evaluated using tester strains of *Salmonella typhimurium*. N-Methyl-N- $\alpha$ -acetoxybenzyl nitrosamine, oxidized on the benzyl moiety, was more toxic and more mutagenic than N-acetoxymethyl-N-benzyl nitrosamine, oxidized on the methyl side chain. This latter compound was weakly toxic and nonmutagenic at the concentrations tested (1-10,000  $\mu$ g/plate). The presence or absence of the *uvrB* repair system had no effect on the toxicity of either compound. Neither the  $\alpha$ -oxidized compounds nor the parent nitrosamine reverted the frame shift mutants. As a mutagen, N-methyl-N- $\alpha$ -acetoxybenzyl nitrosamine was about as active as N-nitroso compounds not requiring metabolic activation, more active than the  $\alpha$ -acetoxydialkyl nitrosamines, and less active than cyclic  $\alpha$ -acetoxy nitrosopyrrolidine. It is suggested that the nature of the metabolic products may aid in determining organotropic carcinogenesis. Methylbenzyl nitrosamine may be oxidized more readily on the benzyl moiety in the esophagus than in

other organs, leading to the formation of large amounts of the proximate carcinogen. This effect may also explain the specificity of other unsymmetrical nitrosamines for the esophagus. (19 refs.)

77-3210 Early Changes in Electrophysiology and Transport of Canine Gastric Mucosa Elicited by a Gastric Carcinogen, N-Methyl-N'-Nitro-N-Nitrosoguanidine (Meeting Abstract). (Eng.) Chou, A. C. (Dept. Physiology, Univ. Texas Medical Sch. Houston, Houston, TX 77025) Kuo, Y.; Shanbour, L. L. *Fed Proc* 36(3): 348; 1977. (no refs.)

77-3211 Acute Effects of N-Methyl-N'-nitro-N-nitrosoguanidine on Canine Gastric Mucosa. (Eng.) Kuo, Y. J. (Dept. Physiology, Univ. Texas Medical Sch. at Houston, 6400 W. Cullen St., Texas Medical Center, Houston, TX 77030) Chou, A. C.; Shanbour, L. L. *J Natl Cancer Inst* 59(1): 131-135; 1977.

The electrophysiological effects of the gastric chemical carcinogen N-methyl-N'-nitrosoguanidine (MNNG) were determined in an in vivo chambered canine stomach and in an in vitro canine gastric mucosal preparation. In the in vivo stomach, the topical application of 2.5 mg MNNG/ml decreased the transmural electrical potential difference, but the systemic blood pressure was essentially unchanged. In the in vitro preparation, exposure of the mucosal side of the isolated canine gastric mucosa to 0.25 and 2.5 mg MNNG/ml for 1 hr sequentially or exposure of the serosal side to 2.5 mg MNNG/ml for 2 hr inhibited net Na<sup>+</sup> and Cl<sup>-</sup> fluxes. With longer duration, the unidirectional fluxes of Na<sup>+</sup> and Cl<sup>-</sup> increased, indicating an increase in permeability. These findings suggest that inhibition of active transport in the gastric mucosa may have an important function in the gastric carcinogenicity of MNNG. (21 refs.)

\* (Rev): 77-3001, 77-3002, 77-3003, 77-3004, 77-3005, 77-3006, 77-3007, 77-3008, 77-3009, 77-3010, 77-3011, 77-3012, 77-3013, 77-3014, 77-3015, 77-3016, 77-3017, 77-3018, 77-3019, 77-3020, 77-3024, 77-3030, 77-3047, 77-3053, 77-3054, 77-3056, 77-3057, 77-3062.

\* (Phys): 77-3218, 77-3219, 77-3229, 77-3230.

\* (Viral): 77-3277.

\* (Immun): 77-3329, 77-3337, 77-3341, 77-3347.

\* (Path): 77-3405, 77-3406, 77-3410, 77-3411, 77-3469, 77-3476, 77-3477, 77-3494.

\* (Epid): 77-3506, 77-3511, 77-3512, 77-3513, 77-3528, 77-3529, 77-3530, 77-3531, 77-3532, 77-3533, 77-3534, 77-3535, 77-3536, 77-3538, 77-3539, 77-3540, 77-3541, 77-3542, 77-3543, 77-3544, 77-3545, 77-3546, 77-3547, 77-3548, 77-3549, 77-3550, 77-3551, 77-3552, 77-3553, 77-3554, 77-3555, 77-3556.



## PHYSICAL CARCINOGENESIS

- 77-3212 **Mutation and Inactivation of Mammalian Cells by Various Ionising Radiations.** (Eng.) Cox, R. (MRC Radiobiology Unit, Harwell, Didcot, Oxfordshire, UK) Thacker, J.; Goodhead, D. T.; Munson, R. J. *Nature* 267(5610): 425-427; 1977.

To determine the relationship between relative biological effectiveness (RBE) and linear energy transfer (LET) values for certain radiations for (1) the induction of mutants deficient in hypoxanthine-guanine phosphoribosyl transferase and (2) cellular inactivation, cultures of freshly isolated human diploid HF19 fibroblasts and established V79 hamster cells were irradiated with helium, boron, or nitrogen ions covering an LET range of 20-470 kiloelectron volts (keV)  $\mu\text{m}^{-1}$ . Plots of the variation of RBE with LET for mutation induction and inactivation in both cell types showed similar humped forms, with maxima in the LET range 85-200 keV  $\mu\text{m}^{-1}$ . In this range, the RBE for mutation induction was about twice that for inactivation. The form of the RBE/LET relationship suggests that mutation and cellular inactivation require that one charged particle should leave either: (1) several energy loss events in a single sensitive cellular component or (2) one energy loss event in each of two targets (such as the 2 strands of a DNA duplex of 2 nanometers diameter). (24 refs.)

- 77-3213 **Chromosome Aberrations in the Leukocytes of Pigs after Half-Body or Whole-Body Irradiation.** (Eng.) McFee, A. F. (Comparative Animal Res. Lab., Oak Ridge, TN 37830) *Mutat Res* 42(3): 395-400; 1977.

Chromosome aberrations were scored in 48-hr WBC cultures from pigs subjected to whole-body or half-body  $\gamma$ -irradiation with 100, 150, 200, 300, or 400 R. Half-body irradiation resulted in the recovery of approx half as many aberrations as did equivalent whole-body exposures at levels of 200 R or less. Higher exposures yielded proportionally fewer anomalies in half-body-irradiated subjects. These lower levels apparently resulted from the selective disadvantage of irradiated cells in coming to mitosis, but they did not seem to be related to the amount of chromosome damage sustained by the cell. When adjustments were made for effective dose to the in vivo cells, the dose-response pattern showed good agreement with published values for mixtures of normal and in vitro-irradiated human lymphocytes. (10 refs.)

- 77-3214 **The Effect of Exposure Rate on Translocation Induction in Somatic and Germ Cells of the**

**Mouse (*Mus musculus*).** (Eng.) van Buul, P. P. (Dept. Radiation Genetics and Chemical Mutagenesis, Univ. Leiden, Wassenaarseweg 72, Leiden, Netherlands) Roos, R. A. *Mutat Res* 42(1): 99-108; 1977.

The effect of exposure rate on translocation induction in the spermatogonia, scored as multivalents in descending spermatocytes, and bone marrow cells of CBA-Rij mice was investigated 65-100 days after x-ray and  $\gamma$ -ray exposures of 400 R. In the range of exposure rates studied, the frequency of chromosomal exchanges in bone-marrow cells decreased from 17.1% at 130 R/min to 4.6% at 0.0287 R/min. In spermatogonia, the frequency dropped from 6.7% at 130 R/min to 1.9% at 0.0287 R/min. The data obtained for both tissues fitted a straight line when plotted against the logarithm of the exposure rate. The ratios between aberration frequencies in bone-marrow cells and spermatogonia, with a mean value of 2.74, were independent of exposure rate. (27 refs.)

- 77-3215 **The Effect of X-Ray Induced Mitotic Delay on Chromosome Aberration Yields in Human Lymphocytes.** (Eng.) Lloyd, D. C. (Natl. Radiological Protection Board, Harwell, Didcot, Oxon, OX11 0RQ, England) Dolphin, G. W.; Purrott, R. J.; Tipper, P. A. *Mutat Res* 42(3): 401-412; 1977.

The extent to which x-ray-induced mitotic delay at 150 and 400 rads influences chromosome aberration yields was examined in human peripheral blood lymphocytes. The dicentric was used as a marker, and aberration yields were obtained for mixed cultures prepared from equal numbers of normal and irradiated cells. The cultures were terminated following incubation times of 36-120 hr. Greater mitotic delay of the order of a few hours was observed at the higher radiation dose. However, most reductions in the numbers of lymphocytes arriving at metaphase by 48 hr were due to interphase death or failure to transform. Analysis of the dicentric distributions that were expected to follow Poisson statistics indicated that cells containing dicentrics were delayed relative to irradiated but aberration-free cells. Cells with one dicentric moved more easily through the first cell cycle than cells containing two dicentrics. Following accidental partial-body irradiation, selection in culture favoring the unirradiated lymphocytes does not distort the aberration yield sufficiently to warrant incubation times in excess of the standard 48-52 hr. (15 refs.)

- 77-3216 **Chromosomal Radiosensitivity and Karyotype in Mice Using Cultured Peripheral Blood Lym-**

lymphocytes, and Comparison with This System in Man. (Eng.) De Boer, P. (Dept. Genetics, Agricultural Univ., Gen. Foulksweg 53, Wageningen, Netherlands) Van Buul, P. P.; Van Beek, R.; Van Der Hoeven, F. A.; Natarajan, A. T. *Mutat Res* 42(3): 379-394; 1977.

The frequencies of x-ray-induced dicentric chromosomes and deletions were studied in cultured human and murine peripheral blood lymphocytes. After doses of 100 and 200 rads, the mouse was as sensitive as man to the induction of dicentrics; the frequency of deletions was higher in the mouse, but the difference was significant only at the 200-rad level. At the 200-rad level, mice with a normal karyotype were compared with mice bearing the T(1;13)70H translocation heterozygote and the Ts(1<sup>13</sup>)70H tertiary trisomic of normal appearance. No differences were found with respect to dicentrics or to deletions. At the 100-rad level, the normal mouse was compared with the tertiary trisomic mouse of the affected phenotype and with the tobacco mouse. The dicentric frequency was significantly higher in the phenotypically abnormal trisomics, but the deletion frequency was higher in the tobacco mice. C-banding of slides enabled breaks to be located in constitutive heterochromatin and euchromatin. When exchanges were classified into three categories, ie, those between eu- and euchromatin, eu- and hetero-, and hetero- and heterochromatin, there was a preference for the first and the last. Few exchanges occurred between chromatins of contrasting type. The equal radiosensitivity of human murine lymphocytes observed here contrasts with earlier data indicating that human lymphocytes are twice as sensitive. Because the discrepancy can be attributed to differences in harvest time of the mouse lymphocytes, the present results do not confirm the so-called "arm number" hypothesis. (29 refs.)

77-3217 **Preleukemic Expression of TL Antigens in X-irradiated C57BL/6 Mice.** (Eng.) Stockert, E. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021) Old, L. J. *J Exp Med* 146(1): 271-276; 1977.

Six-week-old female C57BL/6 mice received 150 R whole-body x-irradiation once weekly for 5 successive wk. Cytotoxicity and absorption studies were conducted over the 53-day postirradiation period. Thymus-leukemia (TL) antigen, which is specified by the TLA locus and is absent in normal C57BL/6 mice, was found in the thymus of 30/49 of the mice before they developed any signs of overt leukemia. Thus, activation of the TLA locus appears to occur during the preleukemic phase of radiation leukemogenesis. A trend toward higher levels of histocompatibility (H-2) alloantigen and reduced levels of Thy-1.2 alloantigen was consistent with preleukemic changes reported for AKR mice and irradiated C57BL mice. Low to moderate levels of MuLV (murine leukemia virus) antigen expression were found on thymocytes of 20/42 irradiated mice. However, no apparent correlation was found between expression of the TL and MuLV antigens. (22 refs.)

77-3218 **Cocarcinogenic Effects of n-Alkanes and Ultraviolet Light on Mice.** (Eng.) Bingham, E. (Dept. Environmental Health, Univ. Cincinnati Medical Center, Kettering Lab., 3223 Eden Ave., Cincinnati, OH 45267) Nord, P. J. *J Natl Cancer Inst* 58(4): 1099-1101; 1977.

A comparison was made of the effects of repeated topical applications to C3H/HeJ mice of three n-paraffins (n-decane, n-dodecane, and n-tetradecane) on the carcinogenic potential of UV light at three wavelength regions: 254, 290-320, and > 350 nm. At 50  $\mu$ l three times per week, all three n-alkanes had a cocarcinogenic effect at 254 nm, but only n-dodecane was effective from 290 to 320 nm. Radiation at wavelengths > 350 nm, generally considered noncarcinogenic, produced tumors on the backs of mice treated with n-decane or n-tetradecane. (9 refs.)

77-3219 **Modulating Factors in the Tumorigenic Effects of Psoralen-Plus-UV in Hairless Mice (Meeting Abstract).** (Eng.) Grube, D. D. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439) Ley, R. D.; Fry, R. J. *Proc Am Assoc Cancer Res* 18: 83; 1977. (no refs.)

77-3220 **Neoplastic Transformation Induced by a Direct Perturbation of DNA (Meeting Abstract).** (Eng.) Tsutsui, T. (Johns Hopkins Univ., Baltimore, MD 21205) Barrett, J. C.; Ts'o, P. O. *Proc Am Assoc Cancer Res* 18: 52; 1977. (no refs.)

77-3221 **DNA Excision Repair in Ultraviolet-irradiated Normal and Malignantly Transformed Mouse Epidermal Cell Cultures.** (Eng.) Bowden, G. T. (NCI, NIH, Building 37, Bethesda, MD 20014) Hohnneck, G.; Fusenig, N. E. *Cancer Res* 37(6): 1611-1617; 1977.

DNA excision repair, as determined by thymine dimer excision and nonsemiconservative DNA synthesis, was measured in differentiating primary cultures of mouse epidermal cells (EPD) and chemically transformed proliferating epidermal cells (PDV). When measured at approximately equal levels of UV-induced damage, the primary cultures of the EPD cells had a reduced excision repair capacity compared to the malignant PDV cells. The EPD cells excised no more than 10% of the original UV-induced dimers in a 24-hr period, but the transformed PDV cells excised 34%. Also, the PDV cells exhibited higher levels of nonsemiconservative repair replication than did the EPD cells at comparable levels of damage. There was no difference in the kinetics of repair replication between the two cell types at a UV level of 10 joules/m<sup>2</sup> over the first 6 hr following irradiation. (41 refs.)



- 77-3222 **The Influence of UV Light on Connective Tissue of Human Skin.** (Ger.) Kreysel, H. W. (Universitäts-Hautklinik Hamburg-Eppendorf, Hamburg, W. Germany) Stermann, W.; Wiskemann, A.; Kimmig, J. *J Soc Cosmet Chem* 28(2): 65-77; 1977.

An increased biosynthesis of proteoglycans and collagen was noted in 30 test subjects exposed, respectively, to UV-A or UV-B radiation. The increase was associated not only with histological and histochemical alterations, but also with modifications of the collagen structure, as demonstrated by immunofluorescence microscopy and collagen atopy. The results suggest that the skin changes caused by solar irradiation, which are identified dermatologically as senile (actinic) elastoses, are an expression of interference with proteoglycan or collagen metabolism of the skin. (74 refs.)

- 77-3223 **The Method of Transformation of the MIRD Absorbed Fractions to Various Physiques** (Meeting Abstract). (Eng.) Yamaguchi, H. (Div. Physics, Natl. Inst. Radiological Sciences 4-9-1, Anagawa, Chiba-shi, Chiba 280, Japan) *Health Phys* 33(2): 157; 1977. (no refs.)

- 77-3224 **Visible Light-Induced DNA Damage in Mouse and Human Cell Culture** (Meeting Abstract). (Eng.) Gantt, R. (NCI, NIH, Bethesda, MD 20014) Ewig, R. A.; Sanford, K. K.; Jones, G. M. *Proc Am Assoc Cancer Res* 18: 204; 1977. (no refs.)

- 77-3225 **Radiation-associated Hyperparathyroidism** (Letter to Editor). (Eng.) LiVolsi, V. A. (Dept. Pathology, Yale Univ. Sch. Medicine, New Haven, CT 06510) Feind, C. R.; LoGerfo, P. *Lancet* 2(8029): 136; 1977.

Twenty-two patients with solitary parathyroid adenomas and synchronous nonmedullary thyroid cancer were investigated for history of radiation exposure. There was no association between the cancers and radiation in this group. (6 refs.)

- 77-3226 **Bomb  $^{14}\text{C}$  and Human Radiation Burden.** (Eng.) Stenhouse, M. J. (Chemistry Dept., Univ. Glasgow, Glasgow, England) Baxter, M. S. *Nature* 267(5614): 825-827; 1977.

The excess human radiation burdens from  $^{14}\text{C}$  produced by nuclear weapons tests were evaluated under the assumption that no radiation dose is biologically harmless. Estimated accumulated absorbed doses due to bomb  $^{14}\text{C}$  were determined

for 30-yr periods ranging from 1954 to 2050. The maximum estimated accumulated dose for soft tissue and bone marrow was 8.81 mrad for 1964-1993; that for bone-lining cells was 6.57 mrad for 1985-2014. The estimated biological damage to reproductive cells from  $^{14}\text{C}$   $\beta$ -irradiation (30-yr accumulated dose) was: 10 single-strand breaks, 1 double-strand break, 0.002-0.24 chromatid break, 0.002-0.01 chromosome break, and 5-15 damaged bases. The combination of two approaches used to determine incidences of zygote mutation per rad of low-dose irradiation administered to the parental generation yielded upper and lower limits of 1,125 and 225, respectively, for the number of individuals who will possess severe deleterious traits attributable to bomb  $^{14}\text{C}$ . The potential radiation burden due to artificially produced  $^{14}\text{C}$  is significant and cannot be dismissed. However, relative to the world population and in view of the time span over which this dose is delivered, these effects will occur unnoticed. (20 refs.)

- 77-3227 **Comparison of Radiosensitivity Between Human Hematopoietic Cell Lines Derived from Patients with Down's Syndrome and from Normal Persons.** (Eng.) Huang, C. C. (Dept. Experimental Biology, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263) Banerjee, A.; Tan, J. C.; Hou, Y. *J Natl Cancer Inst* 59(1): 33-36; 1977.

Seven hematopoietic cell lines, four from the peripheral blood of patients with Down's syndrome (DS) and three from normal persons, were irradiated with 100, 150, 300, and 500 rads from a  $^{60}\text{Co}$  source and harvested for cell count and chromosome studies every 12 hr for 72 hr postirradiation. Cell-growth inhibition and an increase in chromosome aberrations were observed in all cell lines at each dose level and time interval. There was no significant difference in the effects on DS and normal cells. The most common types of aberrations in the 12-hr samples were chromosome and/or chromatid breaks. In later samples, chromatid exchanges predominated. Variance analysis of chromosome aberrations in three DS and three normal lines showed radiation dosage to be the largest component of total variance, following postirradiation duration and cell line. Samples harvested 24 and 36 hr postirradiation generally showed greater effects than samples of other harvest durations. The cell line variance was attributed to the differences among and between individual cell lines rather than differences between DS and normal lines. (24 refs.)

- 77-3228 **The Osteosarcomogenic Effectiveness of the Short-lived  $^{224}\text{Ra}$  Compared with That of the Long-lived  $^{226}\text{Ra}$  in Mice.** (Eng.) Muller, W. A. (Institut für Biologie der Gesellschaft für Strahlen- und Umweltforschung m6H, D-8042 Neuherberg, W. Germany) Luz, A. *Radiat Res* 70(2): 444-448; 1977.

The influence of short-lived  $^{224}\text{Ra}$  on osteosarcomogenesis was evaluated in female NMRI mice.  $^{224}\text{Ra}$  was given in a single injection or in repeated injections, 2x/wk up to 36 wk. Bone tumor incidence increased with dose. Experiments were performed for the following range of parameters: total mean skeletal doses of 30-3,000 rads (1-100  $\mu\text{Ci/kg}$ ), max skeletal dose rates of 6-600 rads/day, and internal irradiation period up to 36 wk. The 1080-rad mean skeletal dose was tested with most variations of the dose-time distribution. An osteosarcoma incidence between 15% and 92% was found that increased with the length of the injection span or, correspondingly, with the lowering of the dose rate. Specific bone tumor production by  $^{226}\text{Ra}$  decreased with an increase in dose. Mice receiving skeletal doses through repeated injections of the short-lived  $^{224}\text{Ra}$  over a long period have a higher bone tumor incidence than mice receiving a single injection of the long-lived  $^{226}\text{Ra}$ . (8 refs.)

77-3229 **Asbestos and Glass Fibres in Bacterial Mutation Tests.** (Eng.) Chamberlain, M. (Medical Res. Council, Pneumoconiosis Unit, Llandough Hosp., Penarth, Glamorgan, Wales) Tarmy, E. M. *Mutat Res* 43(2): 159-164; 1977.

The mutagenicity of asbestos and glass fibers for auxotrophic strains of *Escherichia coli* and *Salmonella typhimurium* in agar plates was studied. The mean particle lengths of the asbestos samples ranged from 1.1 to 3.4  $\mu\text{m}$  and the diameters, from 0.15 to 0.47  $\mu\text{m}$ . The mean particle lengths for code 100 and code 110 glass fibers were 2.7 and 26.0  $\mu\text{m}$ , respectively, and the diameters were 0.12 and 1.0  $\mu\text{m}$ , respectively. No mutagenesis was induced in any bacteria strain by the glass or asbestos fibers, either in the absence or presence of mi-

croosomal enzymes. However, mutations were induced by UV light, potassium chromate, or ethyl methanesulfonate. Because the asbestos and glass fibers were not mutagenic at any of the concentrations used (1-5,000  $\mu\text{g/plate}$ ), the mechanism of asbestos and glass fiber carcinogenesis may not be related to mutagenesis, but rather it may be similar to the unknown mechanism of plastic film carcinogenesis. (24 refs.)

77-3230 **Response of the Differentiated Respiratory Tract Mucosa to Environmental Dusts (Meeting Abstract).** (Eng.) Mossman, B. T. (Univ. Vermont Coll. Medicine, Burlington, VT) Craighead, J. E. *Am J Pathol* 86(2): 70a; 1977. (no refs.)

77-3231 **Elemental Content of Granulomata in Biopsied and Autopsy Lung Tissues (Meeting Abstract).** (Eng.) Brody, A. R. (Univ. Vermont Coll. Medicine, Burlington, VT) Dwyer, D. M.; Vallyathan, N. V.; Craighead, J. E. *Am J Pathol* 86(2): 63a-64a; 1977. (1 ref.)

\* (Rev): 77-3020, 77-3021, 77-3022, 77-3023, 77-3024, 77-3025.

\* (Chem): 77-3108, 77-3122.

\* (Immun): 77-3327, 77-3328.

\* (Path): 77-3367, 77-3372, 77-3398, 77-3430, 77-3452.

\* (Epid): 77-3527, 77-3539.



## VIRAL CARCINOGENESIS

- 77-3232 **Intact Transmission of Avian Leukosis Virus.** (Eng.) Weyl, G. (Dept. Microbiology, Mayo Clinic, Rochester, MN 55901) Dougherty, R. M. *J Natl Cancer Inst* 58(4): 1019-1024; 1977.

Iv inoculation of four age groups of White Leghorn chicks with ALV-F42 ( $2.5 \times 10^4$  infectious U), a Group A field strain of avian leukosis virus, indicated that persistent tolerant infection could be induced as late as 2 wk posthatch, although most birds respond with neutralizing antibody. Contact infection by environmental exposure to ALV was 100% effective in newly hatched or 28-day-old chicks. All contact-infected birds responded immunologically after transient viremia. A follow-up of immune birds from these six groups demonstrated that active replication of ALV continued despite neutralizing antibody. Infectious virus was shed by the oral and cloacal routes, as well as by high vertical transmission from hens to their embryos. Up to  $10^8$  infectious U virus/g feces were shed by 12-day-old viremic chicks; to a lesser extent, virus was also shed in the saliva, as measured by oral washing. The cycle of contact transmission was evaluated by assessing the efficiency of four portals of entry. Exposed skin was most effective in permitting infection, followed by the oral, nasal, and conjunctival routes. (34 refs.)

- 77-3233 **Polypeptides of Endogenous Avian C-Type Viruses: Their Detection in the Plasma Membrane of Normal and Infected Cells.** (Eng.) Kurth, R. (Friedrich Miescher-Laboratorium, Max Planck-Inst., Spemannstrasse 37-39, Tübingen, W. Germany) Bosch, V.; Bolognesi, D. P. *Virology* 78(2): 511-521; 1977.

Avian leukosis sarcoma virus-infected and uninfected fibroblasts from a variety of avian and mammalian species were examined for the cell surface expression of endogenous or exogenous C-type virus polypeptides (VPP). A  $^{14}\text{C}$ -nicotinamide membrane permeability (NAMP) test was used to check cell-surface binding of rabbit antibodies monospecific for individual avian C-type VPP. Most rabbit antisera specific for individual avian RNA-tumor VPP reacted with the surface of cells from a wide variety of infected and uninfected avian species (chicken, jungle fowl, turkey, and ring-necked pheasant). The positive sera included anti-gp85, anti-gp37, anti-p27, anti-p15, and anti-p10. Rabbit anti-p19 and anti-p12 antisera were completely nonreactive on normal cells and reacted only marginally and inconsistently with avian leukosis virus- or avian sarcoma virus-infected chicken cells. Duck, as well as all mammalian cells tested (with one exception), whether infected or uninfected, was negative. The positive

reactions from the uninfected avian cells are interpreted as the result of VPP synthesis by endogenous C-type viruses. A correlation between endogenous VPP expression and positive chicken helper factor activity (previously thought to be due to endogenous synthesis of gp85 and gp37) could not be demonstrated. (47 refs.)

- 77-3234 **The  $\beta$  Subunit of the DNA Polymerase of Avian Sarcoma Virus Strain B77 Is a Phosphoprotein.** (Eng.) Hizi, A. (Dept. Microbiology and Immunology, Duke Univ. Medical Center, Durham, NC 27710) Joklik, W. K. *Virology* 78(2): 571-575; 1977.

The DNA polymerase of B77 avian sarcoma virus was investigated using two techniques: (1) precipitation of DNA polymerase from solubilized virus labeled in vivo with  $^{32}\text{P}$ -orthophosphate by specific antipolymerase antiserum; and (2) purification of the enzyme from virus labeled in vivo with  $^{32}\text{P}$ -orthophosphate. The virus was grown in duck embryo fibroblasts in medium containing  $^{32}\text{P}$ -orthophosphate, and it was isolated from the tissue culture supernatant fluids by centrifugation. In both cases,  $^{32}\text{P}$  was found in bands that coelectrophoresed in polyacrylamide-sodium dodecyl sulfate gels with the  $\beta$  subunit of the DNA polymerase, in the  $\alpha\beta$  and  $\beta$  form of the enzyme. The finding that the  $\beta$  subunit of B77 DNA polymerase is phosphorylated but the  $\alpha$  form, which is probably derived from it by cleavage, is not suggests two alternatives: either the phosphorylation site is on that portion of the  $\beta$  polypeptide chain that is not present in the  $\alpha$  chain, or the cleavage of the  $\beta$  to the  $\alpha$  chain also involves dephosphorylation. (13 refs.)

- 77-3235 **Cell-free Synthesis of Two Proteins Unique to RNA of Transforming Virions of Rous Sarcoma Virus.** (Eng.) Kamine, J. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139) Buchanan, J. M. *Proc Natl Acad Sci USA* 74(5): 2011-2015; 1977.

The 35S RNA of nondefective (nd) Prague B Rous sarcoma virus (RSV) and of transformation-defective (td) Prague B RSV was translated in vitro in a reticulocyte lysate system. A similar spectrum of protein products was found in each case, but two species (molecular wts 25,000 and 18,000 daltons) found in the nd Prague B strain were missing in the td Prague B strain. Neither of these proteins was immunoprecipitated by monospecific antisera against the structural

proteins of avian RNA tumor viruses. Their combined atomic mass of 43,000 daltons corresponds to the amount of genetic coding capacity deleted from the RNA of the td viruses. It is proposed that these proteins are coded for by the putative oncogene (*onc*) or *sarc* (*src*) gene and that one or both of them may be responsible for the oncogenic transformation caused by these viruses in infected cells. (35 refs.)

77-3236    **Phosphorylated and Nonphosphorylated Forms of Avian Sarcoma Virus Polypeptide p19.** (Eng.) Erikson, E. (Dept. Pathology, Univ. Colorado Medical Center, Denver, CO 80262) Brugge, J. S.; Erikson, R. L. *Virology* 80(1): 177-185; 1977.

Two-dimensional electrophoresis of <sup>35</sup>S- and <sup>32</sup>P-labeled Rous sarcoma virus, Prague C strain (PrC), revealed the phosphorylated and nonphosphorylated forms of the major virus protein, p19. Both forms of p19 were immunoprecipitated with antiserum from hamsters bearing Schmidt-Ruppin avian sarcoma virus-induced tumors, supporting previous claims that p19 is a viral RNA-encoded polypeptide. In addition, both forms contained the same methionine-labeled tryptic peptides. Antiserum raised against p27 was used to immunoprecipitate <sup>32</sup>P or <sup>35</sup>S-methionine-labeled polypeptides specifically from extracts of chick embryo fibroblasts. A phosphoprotein that was specifically precipitated from extracts of PrC-infected cells comigrated with a similarly precipitated <sup>35</sup>S-methionine-labeled polypeptide with a molecular wt of approx 76,000 daltons. Moreover, the 76,000-dalton phosphoprotein contained the same tryptic phosphopeptide as p19. The possibility that the binding of p19 to viral RNA depends on the degree of phosphorylation of the peptide is discussed. (16 refs.)

77-3237    **Studies of the Chemical Structure of the Terminal Sequences of Rous Sarcoma Virus RNA and Other Viral RNAs (Meeting Abstract).** (Eng.) Keith, J. M. (Univ. California, Berkeley, CA 94270) *Diss Abstr Int [B]* 37(9): 4441-4442; 1977. (no refs.)

77-3238    **Effects of Age on the Induction of Intracranial Neoplasms in F-344 Rats by Intracerebral Inoculation of Avian Sarcoma Virus (Meeting Abstract).** (Eng.) Copeland, D. D. (Duke Univ. Medical Center, Durham, NC) Bigner, D. D. *Am J Pathol* 86(2): 67a-68a; 1977. (no refs.)

77-3239    **Biochemical and Immunological Characterization of the Major Envelope Glycoprotein of Bo-**

**vine Leukemia Virus.** (Eng.) Devare, S. G. (Lab RNA Tumor Viruses, NCI, Bethesda, MD 20014) Stephenson, J. R. *J Virol* 23(2): 443-447; 1977.

The major envelope glycoprotein of bovine leukemia virus was isolated by lectin-bound Sepharose and DEAE-cellulose column chromatography. This protein has a molecular wt of about 51,000 daltons, similar to that of the major glycoprotein of murine mammary tumor virus, and it lacks detectable immunological cross-reactivity with glycoproteins of other oncornaviruses. Sera from 100% of cattle with clinically diagnosed lymphosarcoma contained high titered antibody to <sup>125</sup>I-labeled bovine leukemia virus glycoprotein, but sera from animals in a disease-free herd were antibody-negative. (20 refs.)

77-3240    **Evaluation of Radioimmunoprecipitation for the Detection of Bovine Leukemia Virus Infection in Domestic Cattle.** (Eng.) Devare, S. G. (Lab. RNA Tumor Viruses, NCI, Bethesda, MD 20014) Chander, S.; Samagh, B. S.; Stephenson, J. R. *J Immunol* 119(1): 277-282; 1977.

The potential application of a recently developed radioimmunoprecipitation test for antibody directed against the major structural protein (p24) of bovine leukemia virus (BLV) was evaluated for use in the detection of BLV infection in domestic cattle. This technique was considerably more sensitive than serologic procedures currently used for this purpose. Radioimmunoprecipitation also had distinct advantages over hematologic criteria, such as those specified by Bendixen's index, for the identification of BLV-infected animals. By radioimmunoprecipitation, high levels of antibody to BLV were demonstrated in the sera of 20 animals with confirmed adult lymphosarcoma. Sera from six calves with lymphosarcoma were negative for BLV antibody, indicating that the calf form of the disease involves etiologic factors distinct from those associated with the adult form. (22 refs.)

77-3241    **Evidence that B-Lymphocytes Carry the Nuclear Pocket Abnormality Associated with Bovine Leukemia Virus Infection: Brief Communication.** (Eng.) Pomeroy, K. A. (Dept. Large Animal Clinical Sciences, Coll. Veterinary Medicine, Univ. Minnesota, St. Paul, MN 55108) Paul, P. S.; Weber, A. F.; Sorensen, D. K.; Johnson, D. W. *J Natl Cancer Inst* 59(1): 281-283; 1977.

Peripheral blood lymphocytes from three cows with persistent lymphocytosis were separated on nylon wool columns into nylon wool-adherent and -nonadherent populations. The percentage of cells coated with surface immunoglobulin (B cells) and the frequency of lymphocytic nuclear pockets in each subpopulation were examined. In each case, the adherent population consisted predominately of B cells with in-



creased nuclear pocket frequency; the nonadherent cells were 98.99% negative for surface immunoglobulin (non-B cells) and contained essentially no nuclear pockets. These findings provide additional evidence that the B subpopulation of cells is highly involved in bovine leukopenia oncogenesis. (30 refs.)

- 77-3242 **Changes Occurring in Bone Marrow Tissue During Initial Stage of Feline Leukemia Virus Infection (Meeting Abstract).** (Eng.) Theilen, G. H. (Univ. California, Davis, CA 95616) Pedersen, N. C.; Lewis, J. P. *Proc Am Assoc Cancer Res* 18: 197; 1977. (no refs.)

- 77-3243 **Expression of Endogenous Retroviral Genes in Leukemic Guinea Pig Cells.** (Eng.) Davis, A. R. (Dept. Microbiology and Immunology, Sch. Medicine, Univ. California at Los Angeles, Los Angeles, CA 90024) Nayak, D. P. *J Virol* 23(2): 263-271; 1977.

The expression of guinea pig retrovirus (TPV), induced by 5-bromodeoxyuridine (BUdR), was studied in guinea pig L<sub>2</sub>C leukemic lymphoblasts by the use of molecular hybridization of viral complementary DNA (cDNA) to cellular RNA. L<sub>2</sub>C leukemic lymphoblasts, leukemic spleen, and BUdR-induced virus-producing cells contained virus-specific RNA: 0.05% (800-960 copies/cell), 0.02% (360 copies/cell), and 0.3% (5,120 copies/cell), respectively. Adult normal liver and spleen, on the other hand, contained < 0.2 copy of viral RNA/cell. Both BUdR-induced cells and L<sub>2</sub>C leukemic lymphoblasts contained 14S, 22S, 35S, and 70S RNA species of total and cytoplasmic virus-specific RNA, as determined by sucrose velocity gradient analysis and hybridization of sucrose gradient fractions to cDNA. Virus-specific mRNA was identified in both BUdR-induced cells and L<sub>2</sub>C leukemic lymphoblasts by the criteria that it cosedimented with purified polyribosomes in a sucrose gradient and changed to a lower sedimentation value if polyribosomes were disaggregated with EDTA prior to centrifugation. Virus-specific messenger RNA (mRNA) obtained from either the polyribosome region of purified polyribosomes or the released messenger region of EDTA-disaggregated purified polyribosomes consisted of 14S, 20S, and 35S species in both BUdR-induced cells and L<sub>2</sub>C leukemic lymphoblasts. Hybridization of cDNA to the RNA of L<sub>2</sub>C leukemic lymphoblasts and BUdR-induced cells was essentially complete. Additionally, leukemic lymphoblast RNA could displace 95% of the hybridization of BUdR-induced GPV 70S RNA to guinea pig DNA. The thermal denaturation midpoints for hybrids formed between GPV cDNA and the RNA of either L<sub>2</sub>C leukemic lymphoblasts or the 70S RNA of BUdR-induced GPV were both 89°C in concentrated (2 times) 0.15 M NaCl plus 0.015 M sodium citrate. These results show that BUdR-induced GPV genes are essentially completely expressed in L<sub>2</sub>C leukemic lymphoblasts and that virus-specific mRNA is present, although the lymphoblasts contain fewer copies of RNA than BUdR-induced cells. (31 refs.)

- 77-3244 **Immunopathogenicity and Oncogenicity of Murine Leukemia Virus. III. Quantitation of Spontaneous Virus Expression.** (Eng.) Croker, B. P. (Dept. Pathology, Duke Univ. Medical Center, Durham, NC 27710) McConahey, P. J.; Murphy, E. D.; Dixon, F. J. *J Natl Cancer Inst* 59(1): 199-205; 1977.

Electron microscopy was used to determine C-type virions in the gut-associated and genital tract epithelia of various mouse strains. The number of morphologically identifiable C-type virus particles varied more than one hundredfold among strains, being high in all strains exhibiting immunologic disease (AKR and NZB) as well as in several immunologically normal strains (LG/J, DBA/2J, NZW) and low in other immunologically normal strains. No relationship was seen between the number of virions found in epithelial and lymphoid tissues. There was, however, a direct correlation between numbers of virions in epithelial tissues and levels of serum gp70. (37 refs.)

- 77-3245 **Leukemogenesis In Vitro Induced by Thymus Epithelial Reticulum Cells Transmitting Murine Leukemia Viruses.** (Eng.) Haas, M. (Dept. Cell Biology, Weizmann Inst. Science, Rehovot 76100, Israel) Sher, T.; Smolinsky, S. *Cancer Res* 37(6): 1800-1807; 1977.

In a study to investigate the role of the thymus in leukemia induction, normal thymus lymphocytes from C57BL/6 mice were cultivated (1) on thymus epithelial reticulum (TER) monolayers from normal mice that did not develop lymphomas or (2) on TER monolayers grown from radiation leukemia virus (RLV)-induced leukemic thymuses. After 3 days cocultivation, 10<sup>6</sup> thymocytes were injected ip into young adult C57BL/6 mice to test for their tumorigenicity. All mice treated with thymocytes cultivated with the TER monolayers from leukemic mice died of disseminated lymphatic leukemia within 3-4 wk. No mice given lymphocytes grown on TER monolayers from normal mice developed lymphomas. The leukemic TER cells produced thymotropic, as well as ecotropic and xenotropic, RLV. Thymocytes that were cultivated on TER from leukemic mice became positive for RLV group-specific antigens, and they produced syncytia in the XC test. It is concluded that the in vitro transformation of the thymocytes grown on TER from leukemic mice is associated with their infection by thymotropic and ecotropic RLV. (27 refs.)

- 77-3246 **Lower Limb Paralysis Induced in Mice by a Temperature-sensitive Mutant of Moloney Leukemia Virus.** (Eng.) McCarter, J. A. (Cancer Res. Lab., Univ. Western Ontario, London N6A 5B7, Canada) Ball, J. K.; Frei, J. V. *J Natl Cancer Inst* 59(1): 179-183; 1977.

A temperature-sensitive mutant of Moloney murine leukemia virus defective in an early function and injected (0.05 ml) into

newborn mice produced lower limb paralysis. The paralysis was due to necrosis of the motor neurons of the spinal cord. It was not associated with budding C-type virus particles on the surfaces of the affected cells. (16 refs.)

- 77-3247 **A Morphological Study on the Ultrastructure and Assembly of Murine Leukemia Virus Using a Temperature-sensitive Mutant Restricted in Assembly.** (Eng.) Yuen, P. H. (Dept. Microbiology, Univ. Illinois, Urbana, IL 61801) Wong, P. K. *Virology* 80(2): 260-274; 1977.

Scanning electron microscope studies were performed on fibroblastic TB cells infected with a temperature-sensitive mutant, ts3, of Moloney murine leukemia virus. Ts3 produced large numbers of both normal particles and multiploids [ie, virions with more than one ribonucleoprotein component (RNP)] in all stages of assembly at the nonpermissive temperature (39°C). In thin sections, budding particles and extracellular immature particles showed the general structural components characteristic of other RNA tumor viruses. Examination of the core shell confirmed that it consisted of polygonal subunits; the double-striated tracks seen in negatively stained virions appeared to be the edges of these subunits, rather than a core membrane. An additional substructure in the core shell extended from the center of each polygonal subunit to the outer periphery of the RNP. The presence of substructures in the form of rings or short tubular substructures in the RNP was consistent with the hypothesis that the RNP is a hollow sphere formed by supercoiling of a single- or double-stranded helix. Multiploids were also observed in ts3-infected cells grown at the permissive temperature and in murine leukemia virus-infected cells. The assembly of both normal particles and multiploids was essentially similar. Multiploids were apparently produced when two or more normal virions assembled in close proximity or in succession at about the same site. The final steps in the assembly of both types of particles were preceded by resealing of the cellular membrane. The laying down of the remaining section of the viral envelope occurred as a separate event. Elongation of the cytoplasmic strands linking the virions to the cell seemed to occur before virion release. The significance of multiploids in mixed infections is discussed. (17 refs.)

- 77-3248 **Mechanism of Formation of Pseudotypes Between Vesicular Stomatitis Virus and Murine Leukemia Virus.** (Eng.) Witte, O. N. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139) Baltimore, D. *Cell* 11(3): 505-511; 1977.

Pseudotypes of vesicular stomatitis virus (VSV) and Moloney murine leukemia virus (MuLV), defined by their resistance to neutralization by anti-VSV antiserum, were released preferentially at early times after infection of MuLV-producing cells with VSV. At later times, after synthesis of MuLV proteins had been inhibited by VSV infection, neither MuLV virions nor the VSV(MuLV) pseudotypes were made. Infection of MuLV-producing cells with mutants of VSV having temperature-sensitive lesions in either G or M protein did not generate pseudotypes at a nonpermissive temperature, indicating that both proteins are needed for pseudotypes to form. Although the pseudotypes resisted neutralization by anti-VSV serum, they were inactivated by anti-VSV serum + complement and precipitated by rabbit anti-VSV serum + goat anti-rabbit IgG. These results, coupled with experiments using a temperature-sensitive mutant of VSV G protein grown at a partly restrictive temperature, suggest that small numbers of VSV G protein are obligately incorporated into VSV(MuLV) pseudotypes. There appears to be a stringent requirement for recognition of the viral core by homologous envelope components as the nucleating step in budding. Only after such a nucleation can the envelope components of the second virus substitute into the membrane of the budding particle. (30 refs.)

- 77-3249 **Murine Leukemia Induced by Viral Ultrafiltrates Negative for XC Plaque Activity.** (Eng.) Correa, J. E. (Seccion Leucemia Experimental, Instituto de Investigaciones Hematologicas, Academia Nacional de Medicina Las Heras 3092, Buenos Aires, Argentina) Basombrio, M. A.; Pasqualini, C. D. *Arch Geschwulstforsch* 46(7): 555-557; 1976.

Filtrates of murine leukemia virus PLLV-T2 were used simultaneously for tissue culture and bioassay, and their leukemogenic effect was tested in BALB mice. It was found that 220-nanometer filtrates were 100% leukemogenic and had a high virus titer ( $10^4$ ) as measured by the XC plaque assay. In contrast, 100-nm filtrates had no plaque forming activity, but were 75% leukemogenic. These results suggest that the viral subunits may be deficient for replication in BALB fibroblasts in vitro, but are able to transform lymphoid cells in vivo. (6 refs.)

- 77-3250 **Exogenous Mouse Interferon in AKR Leukemia (Meeting Abstract).** (Eng.) Roboz, J. P. (Mt. Sinai Sch. Medicine, New York, NY 10029) Ward, M. C.; Holland, J. F.; Bekesi, J. G. *Proc Am Assoc Cancer Res* 18: 198; 1977. (no refs.)

- 77-3251 **Characteristics of MuLV from Malignant and Normal AKR Lymphoid Tissue (Meeting Abstract).** (Eng.) Beardsley, T. R. (Univ. California, Los An-



geles, CA 90024) Haskett, D. R.; Hays, E. F. *Proc Am Assoc Cancer Res* 18: 216; 1977. (no refs.)

- 77-3252 **Splenic Erythroid Response to Friend Polycythemia Virus: Time Course In Vitro after Infection In Vivo.** (Eng.) Hankins, W. D. (Dept. Medicine, Vanderbilt Univ. Sch. Medicine, Nashville, TN 37203) Rosenblatt, P.; Krantz, S. B. *J Natl Cancer Inst* 59(1): 107-111; 1977.

When spleen cells removed from plethoric BALB/c mice shortly after infection with Friend polycythemia virus were cultured, they subsequently increased their rate of Hb synthesis in vitro without addition of the hormone erythropoietin. The increased  $^{59}\text{Fe}$  incorporation into Hb in vitro was part of a well-defined single wave, unlike the progressive increase in Hb synthesis that occurs in vivo. The peak occurred within the same total time (85-105 hr after infection), irrespective of either the virus dose or time after infection when the cells were removed from the animal and cultured. However, the magnitude of the peak increased with an increase in dose over the range 6,250-84,000 focus-forming units and with an increase in the in vivo time from 7 to 24 hr. Experiments in which the medium was varied indicated that the time during which the peak occurred was not artificially determined by depletion of some medium component or the accumulation of an inhibitor. This system may be useful in separating the early events of Friend virus infection from the late effects on erythroid differentiation. (21 refs.)

- 77-3253 **Differentiation, Clonogenicity and Malignancy of Friend Leukemia Cells (FLC) (Meeting Abstract).** (Eng.) Preisler, H. D. (Depts. Medicine A, Roswell Park Memorial Inst., Buffalo, NY 14263) Reese, P.; Rustum, Y. M. *Proc Am Assoc Cancer Res* 18: 103; 1977. (no refs.)

- 77-3254 **Friend Leukemia Cell Line(K-1) Propagating Mainly Spleen Focus Forming Virus (SFFV) (Meeting Abstract).** (Eng.) Ikawa, Y. (Cancer Inst., To-shima-ku, Tokyo 170, Japan) Yoshida, M. *Proc Am Assoc Cancer Res* 18: 215; 1977. (no refs.)

- 77-3255 **Friend Virus Production by Erythropoietin Responsive Cells In Vivo (Meeting Abstract).** (Eng.) Nasrallah, A. G. (Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14203) McGarry, M. P. *Proc Am Assoc Cancer Res* 18: 96; 1977. (no refs.)

- 77-3256 **The Hematopoietic Stem Cell (CFU-S) in Rauscher Leukemia Virus (RLV-A)-Induced Erythroleukemia (Meeting Abstract).** (Eng.) Bergson, A. I. (New York Univ., New York, NY 10003) *Diss Abstr Int [B]* 37(9): 4272; 1977. (no refs.)

- 77-3257 **Inhibitory Action of Neuraminidase of *Vibrio cholerae* in Rauscher Mouse Leukemia.** (Eng.) Barinskii, I. F. (D. I. Ivanovskii Inst. Virology, Acad. Medical Sciences USSR, Moscow, USSR) Kobrinskii, G. D. *Bull Exp Biol Med* 82(11): 1697-1699; 1977.

A mixture of the neuraminidase of *Vibrio cholerae* (50 U/ml) and spleen cells from mice with Rauscher leukemia ( $2.5 \times 10^7$  cells/ml), injected ip into BALB/c mice in a dose of 0.5 ml, markedly inhibited the onset of Rauscher leukemia. At concentrations of 10 and 20 U/ml, however, the enzyme had no such action. Because neuraminidase was nontoxic at all three concentrations, its possible therapeutic action was also investigated. However, ip injection of 0.1 ml of a 50-U/0.1 ml *V. cholerae* neuraminidase solution into mice infected experimentally with Rauscher leukemia had no therapeutic effect. Thus, direct contact between concentrated preparations of neuraminidase and tumor cells may be important. (7 refs.)

- 77-3258 **The Binding of Human Serum Ribonuclease from Hodgkin's Disease Patients with Rauscher Leukemia Virus Reverse Transcriptase (Meeting Abstract).** (Eng.) Bandyopadhyay, A. K. (NCI, Baltimore Cancer Res. Center, Baltimore, MD 21211) Levy, C. C.; Mardiney, M. R. *Proc Am Assoc Cancer Res* 18: 112; 1977. (no refs.)

- 77-3259 **RNA Sequences Specifically Associated with Mouse Intracisternal A Particles.** (Eng.) Lueders, K. K. (Lab. Biochemistry, NCI, Bethesda, MD 20014) Segal, S.; Kuff, E. L. *Cell* 11(1): 83-94; 1977.

The nature of RNA species in isolated intracisternal A particles from mouse myeloma MOPC 104E cells was investigated using complementary DNA (cDNA) made with the polyadenylated RNA of the particles as template. An abundant class of sequences with a genetic complexity of  $2-2.5 \times 10^6$  daltons was found in the cDNA; these sequences represent about 55% of the total cDNA. The entire class of sequences was much more concentrated in tumors of three cell types rich in A particles (myeloma, rhabdomyosarcoma, and neuroblastoma) than in several cell lines apparently devoid of particles. In the MOPC 104E cells, the A-particle-specific sequences comprised almost 8% of the total cytoplasmic polyadenylated RNA. In A-particle-containing fractions of

exponentially growing neuroblastoma cells, the specific sequences were associated principally with RNA molecules sedimenting in the range 26S-32S. There was no detectable homology between the class of specific A-particle sequences detected here and a set of sequences common to several murine C-type viruses. (45 refs.)

**77-3260 Presence of Type C Particles Containing Reverse Transcriptase in L1210 Leukemia. (Eng.)**

Allaudeen, H. S. (Dept. Pharmacology, Yale Univ. Sch. Medicine, New Haven, CT 06510) Bertino, J. R. *J Natl Cancer Inst* 59(1): 227-235; 1977.

A murine L1210 leukemia cell line was grown either in vitro or in vivo by ip injection of  $10^6$  cells/mouse in (C57BL x DBA)F<sub>1</sub> mice. It contained particles that resembled murine leukemia virus. Electron microscopic examination of the cells showed both intracytoplasmic and budding C-type particles. Similar particles were also present in the high-speed pellet (90,000 x g) obtained from both mouse ascitic fluid and the supernatant from cells grown in tissue culture. When the high-speed pellet was centrifuged in a sucrose density gradient, particles banding at densities of 1.16-1.18 g/ml were detected. The virus appeared to be N-tropic. These C-type particles contained an RNA-dependent DNA polymerase (reverse transcriptase) activity, and they performed endogenous DNA synthesis. The enzymes were purified and found to be from the cells and virus particles were purified and found to be indistinguishable in their properties. The molecular wt, as determined by velocity sedimentation and gel filtration, was approx 70,000. Primer template preference studies indicated that the purified enzyme utilized synthetic ribo templates better than synthetic deoxyribo templates, and it could also use a primer template specific for reverse transcriptase. The enzyme could transcribe heteropolymeric regions of 70S RNA from Rauscher murine leukemia virus (R-MuLV), and it had RNaseH activity as well. The enzyme activity was inhibited by antibody directed against the reverse transcriptase from R-MuLV. The biophysical, biochemical, and immunologic properties of the purified enzyme resembled those of the reverse transcriptase from R-MuLV. (40 refs.)

**77-3261 Allelic Structural Genes for the Expression of Mature or Immature Endogenous Type-C Virus in C57BL/He and C57BL/6J Mice. (Eng.)** Boiocchi, M. (Div. Experimental Oncology A, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy) Della Torre, G.; Della Porta, G. *Tumori* 63(2): 163-167; 1977.

C-type particles produced in large numbers by the exocrine pancreas of normal C57BL mice were found by electron microscopy to retain their immature morphology in C57BL/6J mice and to develop into the mature form in C57BL/He

mice. Examination of F<sub>1</sub>, F<sub>2</sub>, and Be<sub>1</sub> hybrids between the two strains indicated a dominant allelism for the immature phenotype. (13 refs.)

**77-3262 Induction of Endogenous Murine Type C Virus by an Arginine Analog: L-Canavanine. (Eng.)**

Aksamit, R. R. (Lab. Immunobiology, NCI, NIH, Bethesda, MD 20014) Long *Virology* 78(2): 567-570; 1977.

The following naturally occurring amino acid analogs were tested for virus induction: S-2-aminoethyl-L-cysteine (lysine), L-canaline dipicrate (lysine), L-canavanine sulfate (arginine), L-homoarginine (arginine), 5-fluoro-D,L-tryptophan (tryptophan), D,L-selenomethionine (methionine), L-ethionine (methionine),  $\beta$ -2-D-L-thienylalanine (phenylalanine), and D,L- $\pi$ -fluorophenylalanine (phenylalanine). A Kirsten sarcoma virus-transformed BALB/3T3 cell was used in the induction experiments. After incubation in induction medium, the compound was removed and the cells were treated with mitomycin C and plated onto normal rat kidney, BALB-3T3, and NIH Swiss embryo monolayers for focus formation. Of the analogs examined, only L-canavanine gave levels of virus induction comparable to those found using cycloheximide. The induction was dependent on canavanine concentration and incubation time, with the greatest induction occurring at 3.6 mM canavanine for 16 hr. Inhibition of protein synthesis alone cannot account for the induction effect of L-canavanine, because in one experiment, cycloheximide inhibited protein synthesis at two concentrations by > 90%, but canavanine and arginine-deficient media inhibited protein synthesis by only 30% and 50%, respectively. Involvement of abnormal canavanine proteins may be critical to the regulation of virus replication. (16 refs.)

**77-3263 Surface Antigens on Transplantable Tumor Cell Lines Producing Mouse Type C Viruses. (Eng.)**

Aoki, T. (Immunology Section, Lab. Viral Carcinogenesis, NCI, NIH Public Health Service, U.S. Dept. Health, Education, and Welfare, Bethesda, MD 20014) Herberman, R. B.; Hartley, J. W.; Liu, M.; Walling, M. J.; Nunn, M. *J Natl Cancer Inst* 58(4): 1069-1079; 1977.

The presence of murine C-type viruses and virus-associated antigens was investigated in a variety of transplantable mouse tumor lines induced by x-irradiation, benzpyrene, methylcholanthrene, mineral oil, Rauscher murine leukemia virus, and Gross murine leukemia virus. The type of virus isolated and antigens detected could not be correlated with the original method of tumor induction, but testing of most tumor lines for infectious virus at various levels of in vivo or in vitro passage yielded isolates that were consistent in tissue culture host range for each tumor. In contrast, during in vivo transplantation, some of the lines underwent a considerable



change in the pattern of virus-associated cell-surface antigens. Upon retransplantation and passage of the cultured cells in mice, the surface antigens gradually returned to the original in vivo patterns and occasionally acquired additional C-type virus-associated antigens not detected in the original tumor line. To test for association of antigens with infectious virus, appropriate tissue culture cell lines were infected with the viruses isolated from the tumors. In these infected indicator cells, some new virus-associated cell-surface and virion envelope antigens were detected, but the complete array of antigens found in the original tumor lines was not acquired. These findings indicate the presence of several different C-type viruses in long-transplanted cell lines and demonstrated that environment and host cell factors may have major influences on the expression of virus-associated antigens. (47 refs.)

- 77-3264 **Structural Antigens on the Surface of Type-C Virus and on Virus-Infected Cells (Meeting Abstract).** (Eng.) Kende, M. (NCI, NIH, Bethesda, MD 20014) Oroszlan, S.; Donahoe, R.; Kelloff, G. *Proc Am Assoc Cancer Res* 18: 101; 1977. (no refs.)

- 77-3265 **P-30 Antigen in Mouse Prostate.** (Eng.) Kind, P. (Dept. Microbiology, George Washington Univ. Sch. Medicine, Washington, DC 20037) *Cancer Treat Rep* 61(2): 129-130; 1977.

Prostate tissue extracts were obtained from male BALB/c mice who were sacrificed 9 wk after castration or sham castration at 21-22 days of age. Using microcomplement fixation and rocket immunoelectrophoresis techniques, p30 antigen of C-type viruses was found in the tissues of the castrated animals. However, little or no activity could be found in sham-castrated mice. Neither of the two immunologic methods proved adequate for quantitative assays. (13 refs.)

- 77-3266 **Induction of C-Type Endogenous Murine Virus. Tentative Isoelectric Point Discrimination of the p30 Polypeptide, and Aspects of Cell Transformation.** (Fre.) Laprevotte, I. (Laboratoire d'Hématologie expérimentale, U.E.R. d'Hématologie, Paris-VII, Hôpital Saint-Louis, 2, place du Docteur-Fournier, 75010 Paris, France) Chuat, J. C.; Bernard, C.; Canivet, M.; Pilon, C. *C R Acad Sci [D] (Paris)* 284(17): 1737-1740; 1977.

Endogenous ecotropic and xenotropic murine C-type viruses induced in K-BALB-3T3 cells treated with iododeoxyuridine were isolated by infection of the appropriate indicator cells (mouse C<sub>3</sub>H, 3T3-FL, BALB-3T3; rat NRK; mink Mv-1-Lu; rabbit SIRC). The isoelectric point of the major viral poly-

peptide (molecular wt 30,000) was 6.1 (minor peaks around 5.6 and 6.6) for the ecotropic virus and 5.7 (secondary peak around 5.4) for the xenotropic virus. Morphological transformation was observed in the C<sub>3</sub>H, 3T3-FL, NRK, and BALB-3T3 cells, but not in the Mv-1-Lu and SIRC cells. (19 refs.)

- 77-3267 **Two Species of Type C Viral Core Polypeptide on AKR Mouse Leukemia Cells.** (Eng.) Tung, J. S. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021) Pinter, A.; Fleissner, E. *J Virol* 23(2): 430-435; 1977.

Two species of glycosylated C-type viral core polypeptide (gp85 and gp95) were identified on the surface of AKR spontaneous leukemia cells. One of these cell surface polypeptides was shown by immunoprecipitation to have the p30, p15, p12, and p10 antigenic determinants of murine leukemia virus; the other had murine leukemia virus p30, p15, and p12, but not p10, determinants. Both species were also expressed on thymocytes from 6-mo-old preleukemic AKR mice. (21 refs.)

- 77-3268 **Polypeptides of Cells Transformed by RNA or DNA Tumor Viruses.** (Eng.) Strand, M. (Dept. Pharmacology and Experimental Therapeutics, Johns Hopkins Univ., Sch. Medicine, Baltimore, MD 21205) August, J. T. *Proc Natl Acad Sci USA* 74(7): 2729-2733; 1977.

Cell transformation by RNA or DNA tumor viruses resulted in a complex change in the type, concentration and/or properties of approx 30% of the cellular polypeptides. Most of these changes were the same regardless of whether the virus was RNA or DNA. The identities of many of these polypeptides remain to be determined. (26 refs.)

- 77-3269 **Definition and Characterization of the Leukemia- and Sarcoma-Specific RNA Components of Murine RNA Tumor Viruses (Meeting Abstract).** (Eng.) Maisel, J. E. (Univ. California, Berkeley, CA 94720) *Diss Abstr Int [B]* 37(9): 4325-4326; 1977. (no refs.)

- 77-3270 **Cyclic AMP Induced Morphological Transformation of Rat Cell Infected with Mouse Sarcoma Virus (Meeting Abstract).** (Eng.) Somers, K. D. (Eastern Virginia Medical Sch., Norfolk, VA 23501) *Proc Am Assoc Cancer Res* 18: 101; 1977. (no refs.)

**77-3271 MSA and EGF Receptors on Sarcoma Virus Transformed Cells and Human Fibrosarcoma Cells in Culture.** (Eng.) Todaro, G. J. (Lab. Viral Carcinogenesis, NCI, NIH, Bethesda, MD 20014) De Larco, J. E.; Nissley, S. P.; Rechler, M. M. *Nature* 267(5611): 526-528; 1977.

The binding of two polypeptide growth factors that are efficient stimulators of cell DNA synthesis, epidermal growth factor (EGF) and multiplication stimulating activity (MSA), to different types of transformed cells was investigated. Transformation of normal rat kidney (NKR clone 2) and mink lung cells with either murine or feline sarcoma viruses abolished approximately 90% of the specific EGF binding, but did not significantly reduce MSA binding. Thus sarcoma virus transformation selectively perturbs the EGF receptor system. In two human fibrosarcoma cell lines, 8387 and HT10180, MSA binding was selectively lost but EGF bound to the same extent as normal fibroblasts. This selective loss or unavailability of MSA receptors in the fibrosarcoma cell lines suggests that an alteration in the MSA-receptor system may have had a role in cellular transformation. These cell lines may produce MSA-like polypeptides that could occupy MSA receptors to stimulate uncontrolled cell division. (20 refs.)

**77-3272 Differences in Mouse Mammary Tumor Viruses: Relationship to Early and Late Occurring Mammary Tumors.** (Eng.) Schlom, J. (NCI, Bethesda, MD 20014) Colcher, D.; Drohan, W.; Kettmann, R.; Michaelides, R.; Vlahakis, G.; Young, J. *Cancer (Suppl)* 39(6): 2727-2733; 1977.

Inbred mouse strains have been classically categorized into (1) high-incidence mammary carcinoma strains with tumors occurring relatively early in the life (C3H, RIH, and GR) or (2) low- or moderate-incidence strains with tumors occurring later in life (C3HfC57BL and BALB/c). Differences in the RNA genome of the mouse mammary tumor virus (MMTV) from these strains were examined by molecular hybridization of radioactively labeled viral 60S-70S RNA's with radioactively labeled complementary DNA's. The results show that the early mammary tumors that occur at high frequency contain recycled or MMTV-S-specific sequences in their DNA; these sequences are not found in the DNA of the tumors that occur at lower frequency and later in life. DNA from mammary tumor cells and normal liver cells of GR mice both contained sequences related to the recycled MMTV-S sequences, indicating that the MMTV of GR mice is transmitted as a germinal provirus. In the other strains, a non-germ line transmission is clearly demonstrated. (23 refs.)

**77-3273 Levels of MMTV Sequences in DNA of "Virus-" and "Hormone-Induced" Malignant**

**Mammary Epithelial Cells of the BALB/c Mouse (Meeting Abstract).** (Eng.) McGrath, C. M. (Dept. Biology, Michigan Cancer Foundation, Detroit, MI 48201) Marineau, E. J.; Voyles, B. A. *Proc Am Assoc Cancer Res* 18: 245; 1977. (no refs.)

**77-3274 Murine Mammary Tumor Virus Infection of Mouse Mammary Epithelial Cells In Vitro (Meeting Abstract).** (Eng.) Vaidya, A. (Inst. Medical Res., Camden, NJ 08103) Lasfargues, E. Y. *Proc Am Assoc Cancer Res* 18: 240; 1977. (no refs.)

**77-3275 Experimental Infection of Non-Murine Cells by Murine Mammary Tumor Virus (Meeting Abstract).** (Eng.) Howard, D. K. (Meloy Lab. Inc., Springfield, VA 22151) Colcher, D. M.; Teramoto, Y. A.; Young, J. M. *Proc Am Assoc Cancer Res* 18: 108; 1977. (no refs.)

**77-3276 Studies on Adenovirus Type 9-induced Mammary Fibroadenomas in Rats and Their Malignant Transformation.** (Eng.) Jonsson, N. (Inst. Pathology, Univ. Hosp., S-221 85 Lund, Sweden) Ankerst, J. *Cancer* 39(6): 2513-2519; 1977.

Mammary fibroadenomas developed in 27/27 female W/Fu rats inoculated when newborn with adenovirus type 9 (Ad 9) administered sc on the back (0.3-0.5 ml) or sc and ip (0.2 + 0.2 ml). The latency period was 14-25 wk. No tumors were seen in 24 Ad 9-inoculated males or in 16 rats of both sexes infected with Ad 5. After 3-14 mo, malignant transformation of the tumor stroma resulted in different types of sarcoma in three rats: round-cell liposarcoma, osteosarcoma, and fibrosarcoma plus malignant mesenchymoma. In another animal the stroma was highly cellular, suggesting a transition to fibrosarcoma. Malignant transformation of the tumor epithelium was not observed. Tumor cells contained Ad 9-specific T antigen, and rats with transplanted tumors were immunized to T antigen. Mammary fibroadenomas without signs of malignant transformation developed in 8/9 female rats inoculated with Ad 9 as adults. Neither neonatal thymectomy nor total body x-irradiation (300 R) significantly shortened the induction time of the virus-induced fibroadenomas or increased the frequency of malignant transformation in females. However, one lipoma and one highly differentiated liposarcoma appeared in two male rats. The results provide an example of the progression of a benign virus-induced tumor into a malignant neoplasm. (10 refs.)



- 77-3277 **Viral Genome Integration in Syrian Hamster Cells After Chemical and Virus (Meeting Abstract).** (Eng.) DiPaolo, J. A. (NCI, Bethesda, MD 20014) Casto, B. C.; Miyagi, M.; Popescu, N. C. *Proc Am Assoc Cancer Res* 18: 94; 1977. (no refs.)

- 77-3278 **Induction of Subcutaneous Sarcomas in the Progeny of Syrian Golden Hamsters After Treatment with Adenovirus 12 During Pregnancy.** (Ger.) Ivankovic, S. (Institut für experimentelle Toxikologie und Chemotherapie am Deutschen Krebsforschungszentrum, Im Neuenheimer Feld 280, D-6900 Heidelberg, W. Germany) *Z Krebsforsch* 88(3): 323-325; 1977.

Sc polymorphic sarcomas were induced in 8/27 offspring of Syrian golden hamsters treated on day 15 of gestation with adenovirus 12. This is the first example of tumor induction by prenatal exposure to oncogenic viruses. (4 refs.)

- 77-3279 **Promotion of Incidence of Adenovirus Type 12 Transplantable Tumors by Carrageenan, a Specific Antimacrophage Agent.** (Eng.) Lotzova, E. (Dept. Developmental Therapeutics, M. D. Anderson Hosp., Univ. Texas System Cancer Center, Texas Medical Center, Houston, TX 77030) Richie, E. R. *J Natl Cancer Inst* 58(4): 1171-1172; 1977.

Carrageenan, a sulfated polygalactose with known macrophage-toxic properties, was used to determine the role of macrophages in resistance to adenovirus type 12 transplantable tumors. A single ip injection of 5 or 10 mg carrageenan led to an increased incidence and more rapid growth of tumors in C3H mice. Carrageenan was most effective if given 1 day before tumor inoculation; the effectiveness decreased with increasing intervals before or after inoculation. The macrophage stabilizer poly-2-vinylpyridine N-oxide, injected sc (150 mg/kg) 1 day before carrageenan administration, reduced the tumor incidence. These data support the importance of macrophages in tumor immunity. (15 refs.)

- 77-3280 **Persistent Adenovirus Infections of Nonpermissive Monkey Cells.** (Eng.) Baum, S. G. (Dept. Medicine and Cell Biology, Albert Einstein Coll. Medicine, Bronx, NY 10461) *J Virol* 23(2): 412-420; 1977.

Persistent infections of monkey cells were established by the use of human adenovirus 2 and 7. The persistently infected cells show no morphological changes, but continue to produce low titers of infectious adenovirus. The inapparent infection can, at any time, be converted to a cytolytic productive one by superinfection with simian virus 40. Persistence in this

system does not appear to result from multiple rounds of lytic infection, nor is it mediated by production of defective interfering particles. The persistently infected cells do not possess the characteristics of oncogenic transformation. The results of these studies also show that the nonpermissiveness of monkey cells to adenovirus replication can be partially overcome by infection at high multiplicity. (28 refs.)

- 77-3281 **One Predominant 5'-Undecanucleotide in Adenovirus 2 Late Messenger RNAs.** (Eng.) Gelinas, R. E. (Cold Spring Harbor Lab., Cold Spring Harbor, NY 11724) Roberts, R. J. *Cell* 11(3): 533-544; 1977.

Oligonucleotides containing the 5' termini of adenovirus 2 messenger RNA (mRNA) were selectively retained on columns of dihydroxyboryl cellulose. When total late adenovirus 2 mRNA was treated with RNAase T1, a single 5' terminal oligonucleotide was isolated, although in several states of methylation. This oligonucleotide has the general structure m<sup>7</sup>G<sup>5'</sup> ppp<sup>5'</sup> AmCmU(C,U)G. Since at least 12 individual species of mRNA must be present late after infection, this finding was unexpected. Its significance is discussed. (36 refs.)

- 77-3282 **A Histone-like Protein from Adenovirus Chromatin.** (Eng.) Lischwe, M. A. (Dept. Chemistry and Biochemistry, Southern Illinois Univ., Carbondale, IL 62901) Sung, M. T. *Nature* 267(5611): 552-554; 1977.

The distribution of basic residues in polypeptide VII, a major core protein found in adenovirus (mass ratio of VII:DNA is 1:1), was compared to that of histones, which have a similar ratio in eukaryotic chromatin. Sequence analysis showed that the first 27 residues at the amino end of VII are enriched in basic amino acids, correlating with the highly basic end in histones that has been implicated in DNA binding. The similarity of the cationic residue distribution in both polypeptide VII and the arginine-rich histone H<sub>4</sub> suggests that the basic region in VII may also serve as a DNA combining site. Endoprotease processing of a 20-residue fragment from the amino end of Pro VII, a precursor of VII, may enable this region to regain its binding capacity and, thus, allow assembly of adenovirus chromatin. (20 refs.)

- 77-3283 **DNA Polymerases in Adenovirus Type 5-infected and Uninfected KB Cells. Induction of an  $\alpha$ -Type DNA Polymerase in Adenovirus Type 5-infected and in Fast Growing Cells.** (Eng.) de Jong, A. (Lab. Physiological Chemistry, State Univ., Utrecht, Netherlands) Van

Der Vliet, P. C.; Jansz, H. S. *Biochim Biophys Acta* 476(2): 156-165; 1977.

DNA polymerase activities in uninfected human KB cells or KB cells infected with adenovirus type 5 (Ad 5) were compared to investigate the possible existence of a viral-induced or modified DNA polymerase during Ad 5 DNA replication. Upon DNA-cellulose chromatography, three components were found in both infected and uninfected cells; the major component contained DNA polymerase  $\alpha$ . Two minor components were also found, one that did not bind to DNA-cellulose and one that bound strongly. The latter component contained DNA polymerase  $\beta$ . No difference in properties between uninfected or Ad5-infected KB cells was found for the  $\beta$ -polymerase. Two forms of DNA polymerase  $\alpha$  ( $\alpha$ I and  $\alpha$ II) were found after DEAE-cellulose chromatography. DNA polymerase  $\alpha$ II was the main activity in the material obtained from infected cells, and DNA polymerase  $\alpha$ I was the main activity in stationary uninfected cells. Fast-growing uninfected cells, however, contained DNA polymerase  $\alpha$ II as the main activity, suggesting that DNA polymerase  $\alpha$ II is related to the level of DNA synthesis rather than to the presence of Ad5 virus. No evidence for a new viral-induced DNA polymerase was found. (30 refs.)

An Epstein-Barr virus (EBV) genome-positive lymphoma was discovered in an Israeli Arab child, and the lymphoma cells were established in continuous culture. The 4-yr-old boy developed four tumors in the mandibula and maxillae, on both sides, in 1 wk. A huge abdominal mass was also palpated. Biopsy of the mandibular tumor revealed a classical histologic picture of Burkitt's lymphoma. Forty-eight hours after the start of Cytosin therapy (40 mg/kg), the tumors had greatly diminished in size, and after a week they could not be seen or palpated. Tumors that appeared on two subsequent occasions were treated by surgery and chemotherapy. The presence of the EBV genome in the biopsy material was demonstrated by nucleic acid hybridization, and 50%-60% of the cells were positive for Epstein-Barr nuclear antigen (EBNA) by immunofluorescence. The lymphoma line (LB-132) established from the patient's tumor tissue also carried the EBV genome, and it was EBNA-positive at the earliest passage examined (4th-6th); 1%-3% of the cells were positive for viral capsid antigen. The similar histology, cytology, and cytogenetics of EBV-negative and EBV-positive lymphomas suggest that their final evolution to autonomous neoplasia may be similar, although the original transformation event may have been caused by different viruses in the two types. (32 refs.)

77-3284 **Antibodies to Epstein-Barr Virus in Nasopharyngeal Carcinoma and Other Neoplastic Conditions.** (Eng.) Kottaridis, S. D. (Hellenic Anticancer Inst., Papanikolaou Res. Center Oncology and Experimental Surgery, 171 Alexandras Ave., Athens 603, Greece) Dafnou, M.; Besbeas, S.; Garas, J. *J Natl Cancer Inst* 59(1): 89-91; 1977.

Sera from 23 patients with nasopharyngeal carcinoma (NPC), 69 patients with benign breast neoplasms, 125 with adenocarcinoma of the breast, and 79 healthy controls were examined for Epstein-Barr virus (EBV) antibodies. The percentage of positive sera differed among the groups studied. High-titer antibody levels were observed in the NPC group, but no statistical difference was found among the other groups of patients and controls. The NPC patients had geometric mean titers of 640 and 35 for EBV viral capsid antigen and early antigen, respectively. The data reaffirm the association of EBV with NPC but do not support its etiologic role in the development of other human neoplasms. (22 refs.)

77-3285 **A Case of an Epstein-Barr Virus (EBV) Genome-carrying Lymphoma in an Israeli Arab Child.** (Eng.) Goldblum, N. (Chanock Centre Virology, Hebrew Univ. Hadassah Medical Sch., Jerusalem, Israel) Ben-Bassat, H.; Mitrani, S.; Andersson-Anvret, M.; Goldblum, T.; Aghai, E.; Ramot, B.; Klein, G. *Eur J Cancer* 13(7): 693-698; 1977.

77-3286 **A New Class of Infectious Agents Detectable by the Production of Chorioallantoic Membrane Lesions by Human Lymphoblastoid Cell Lines and Their Culture Supernatants.** (Eng.) Longenecker, B. M. (Dept. Immunology, 845 Medical Sciences Building, Univ. Alberta, Edmonton, Alberta T6G 2H7, Canada) Menezes, J.; Sanders, E. J.; Pazderka, F.; Ruth, R. F. *J Natl Cancer Inst* 58(4): 853-862; 1977.

The iv injection of  $2.5 \times 10^5$  cells and their tissue culture supernatants (CS) from human lymphoblastoid cell lines (LCL) induced lesions on the chorioallantoic membrane (CAM) of chick embryos. Injection of cells and CS from non-LCL and normal human lymphocytes induced few or no lesions. Irradiated chick embryos were more sensitive to lesion formation than were nonirradiated embryos. The  $\log_{10}$  CAM lesions induced in irradiated (500 rads) embryos were a linear function of the  $\log_{10}$  cells (from LCL) in the inoculum; the slope was 1.0, within experimental error. Lesion formation did not depend on the presence of Epstein-Barr virus (EBV), since lesions were also induced by cells and extracts derived from EBV genome-free LCL. Lesion-inducing activity associated with CS was filterable through 0.22- $\mu$  filters, it sedimented at 78,000 x g and it was sensitive to heat inactivation (56 C for 30 min), UV irradiation, chloroform, sera from chicks immunized against CS, and certain human sera. Lesion-inducing activity associated with cells and extracts was resistant to 5,000 rads of  $\gamma$ -radiation. B<sup>2</sup>/B<sup>3</sup> embryos (the B locus is the major histocompatibility locus of chickens) were more sensitive to lesion formation than were B<sup>15</sup>/B<sup>21</sup> and outbred embryos. The data suggest that the irradiated chick em-



bryo may be useful in the detection of unidentified infectious agents associated with human leukemia and lymphoma. (26 refs.)

- 77-3287 The Use of Temperature Sensitivity and Selective Cell Culture Systems for Differentiation of Herpes Simplex Virus 1 and 2 in a Clinical Laboratory.** (Eng.) Nordlund, J. J. (Veterans Admin. Hosp., West Haven, CT 06516) Anderson, C.; Hsiung, G. D.; Tenser, R. B. *Proc Soc Exp Biol Med* 155(1): 118-123; 1977.

A combination of temperature sensitivity plus microplaque formation in chick embryo (CE) cell monolayers was used to differentiate between herpes simplex virus (HSV) types 1 and 2 in order to assess their routine use in a diagnostic virology laboratory. The virus samples, obtained from patients with herpetiform lesions, were placed directly into a rabbit kidney (RK) cell culture tube. Forty new isolates were tested for microplaque formation in CE cells and for temperature sensitivity. All HSV-2 isolates produced distinct microplaques in CE cells, but the newly isolated HSV-1 strains failed to do so. HSV-1 strains showed similar or slightly lower titers in RK cells when incubated at 35 or 40 C, but the HSV-2 isolates showed distinctly lower titers or delayed cytopathic effects at 40 C. Virus typings were confirmed by neutralization and/or immunofluorescence tests. These results indicate that the use of a biological marker (formation of plaques by HSV-2 in CE cells) and a temperature marker (resistance of HSV-1 at 40 C) provides a rapid, convenient, and economical method for distinguishing HSV-1 from HSV-2 that can be applied routinely in a clinical laboratory. (17 refs.)

- 77-3288 Interference Between Strains of Type 1 and Type 2 Herpes Simplex Virus.** (Eng.) Purifoy, D. J. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77030) Powell, K. L. *Virology* 77(1): 84-94; 1977.

Herpes simplex virus type 2 (HSV-2, 10 and 100 plaque-forming units/cell) markedly interfered with the replication of herpes simplex virus type 1 (HSV-1, 10 plaque-forming units/cell) upon simultaneous infection of HEp-2 cells. The interference occurred even when HSV-2 was added 3 hr after HSV-1, indicating that interferon was not involved and that interference did not occur at the adsorption, penetration, or uncoating stage. UV irradiation destroyed the interfering capacity of HSV-2. The HSV-1 progeny of the mixed infections were phenotypically mixed. In most cases, prior infection with homologous strains slightly inhibited the replication of a superinfecting HSV-1 strain. An exception to this finding is HSV-1 strain MP, whose replication was accelerat-

ed by prior infection with nondefective HSV-1 strains. The implications of these findings to studies of defective HSV, studies of the genetic interactions between HSV-1 and HSV-2, and to clinical infections with HSV are discussed. (14 refs.)

- 77-3289 Biochemical Transformation of Mouse Cells by Fragments of Herpes Simplex Virus DNA.** (Eng.) Maitland, N. J. (Cold Spring Harbor Lab., Post Office Box 100, Cold Spring Harbor, NY 11724) McDougall, J. K. *Cell* 11(1): 233-241; 1977.

Unique DNA fragments derived from herpes simplex virus 2 (HSV-2) strain 333 by cleavage with the restriction endonucleases Eco RI and Hind III and random DNA fragments derived by mechanical shearing were used to transform mouse L cells lacking the enzyme thymidine kinase (TK-). Selection in HAT medium established clones of cells with a TK+ phenotype. The biochemical properties of the TK activity in the cytoplasm of the transformed cells showed it to be of viral origin. The transformed cells also exhibited HSV-specific immunofluorescence. It was further demonstrated that the location of the TK gene is in the long segment of the HSV-2 genome (the L region), probably within 35 units of the internal and terminal redundant sequences, between map units 53.2 and 64.6. (32 refs.)

- 77-3290 Comparative Analysis of Polypeptides Induced by Type 1 and Type 2 Strains of Herpes Simplex Virus.** (Eng.) Powell, K. L. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77030) Mirkovic, R.; Courtney, R. J. *Intervirology* 8(1): 18-29; 1977.

The polypeptides produced by seven type 1 and eight type 2 strains of herpes simplex virus were analyzed by polyacrylamide gel electrophoresis. The results demonstrated little variation in the strains of a given type, but the polypeptides for each type were clearly different from those produced by the other type. (20 refs.)

- 77-3291 Isolation of High-molecular-weight Infectious DNA from Type 1 Herpes Simplex Virus.** (Eng.) Filatov, F. P. (D.I. Ivanovskii Inst. Virology, Acad. Medical Sciences USSR, Moscow, USSR) Manykin, A. A.; Monastyreva, L. A. *Bull Exp Biol Med* 83(1): 28-31; 1977.

A technique is presented that can be used to isolate high molecular wt DNA molecules of the L2 strain of herpes simplex virus type 1. If infectivity is used as a measure of the nativeness of the molecule, then this technique yields native molecules. (13 refs.)

- 77-3292 **Cell-free Synthesis of Herpes Simplex Virus Proteins.** (Eng.) Cremer, K. J. (Dept. Therapeutic Radiology, Yale Univ. Sch. Medicine, New Haven, CT 06510) Summers, W. C.; Gesteland, R. F. *J Virol* 22(3): 750-757; 1977.

Polyribosomes isolated from mouse LMTK- cells infected with herpes simplex virus type 1 (HSV-1) were used to program the cell-free translation of  $^{35}\text{S}$ -methionine-labeled amino acids. The incorporation of  $^{35}\text{S}$ -methionine into the polypeptides, with apparent molecular wts of 25,000 to 160,000 on electrophoresis in sodium dodecyl sulfate-polyacrylamide gels, were synthesized by wild-type HSV-infected polyribosomes. Polyribosomes prepared from thymidine kinase-negative HSV mutants directed the synthesis of three putative nonsense termination polypeptides. The HSV-specific polypeptides synthesized in vitro were precipitated with antiserum to HSV-infected cell proteins. This translation system will allow study of the nature of the mutations in the HSV thymidine kinase gene; it may also provide useful substrates for studies of posttranslation modification reactions. (22 refs.)

- 77-3293 **Sites of Integration of Herpes Simplex Virus (HSV) Thymidine Kinase (TK) Gene in Transformed Human Cells (Meeting Abstract).** (Eng.) Donner, L. (Baylor Coll. Medicine, Houston, TX 77030) Kit, S. *Proc Am Assoc Cancer Res* 18: 171; 1977. (no refs.)

- 77-3294 **Scanning Electron Microscopic Studies of Herpes Simplex Virus Transformed Cells.** (Eng.) Rossowski, W. (Inst. Nuclear Res., Warsaw, Poland) Komitowski, D.; Darai, G.; Munk, K. *Oncology* 34(1): 1-5; 1977.

The surface morphology of rat embryo fibroblasts (REF cells) and REF cells transformed by herpes simplex virus (REF-Tep-NP and REF-Tsp-NP cells) in exponentially growing and density-inhibited cultures was examined by scanning electron microscopy. Contact-inhibited normal REF cells appeared as polygonal or spindle-shaped cells having a smooth surface, with only a few curving, filamentlike unbranched cellular microvilli. The cell surface of the REF-Tep-NP cells was characterized by a smooth appearance, with a few rounded blebs or ruffled membranes. Cellular microvilli were markedly reduced, even at the exponentially growing phase. In contrast, REF-Tsp-NP cells in the early G<sub>1</sub> phase of the cell cycle (density-inhibited) had a significant percentage of their membrane in the form of microvilli. In exponentially growing REF-Tsp-NP cells, most of the cell surface formed numerous folded villi, many of which were

branched. C-type virus particles budding from the cell surface of the transformed cells were commonly seen. Virus-induced cytopathic effects observed by scanning electron microscopy included rounding up and detachment of degenerating cells from the substrate. (14 refs.)

- 77-3295 **Investigations of the Evidence of Herpes Simplex Virus Type 2 (HSV-2) Antibodies in Patients with Carcinoma of the Cervix.** (Ger.) Dostal, V. (Institut für Krebsforschung, Universität Wien, Borschkegasse 8a, A-1090 Vienna, Austria) Fanta, D.; Reiss-Gutfreund, R. J.; Janisch, H.; Berger, R. *Wien Klin Wochenschr* 89(6): 201-203; 1977.

Neutralizing antibodies to herpes simplex virus types 1 and 2 (HSV-1 and HSV-2) were determined in the sera of 126 patients. Infection was detectable in nearly 100% of the cases in each of the three groups investigated: patients with cervical carcinoma, women with chronic recurrent HSV infection in the genital area, and women (controls) without any history of HSV infection. The percentage of subjects with HSV-2 antibodies was significantly higher in the cervical carcinoma patients (38%) than in the controls (12%). The results are compared with the findings of other investigators, and the possible etiological role of HSV in cancer is discussed. (20 refs.)

- 77-3296 **Partial Characterization of a Herpes-Type Virus (K9V) Derived from Kaposi's Sarcoma.** (Eng.) Glaser, R. (Dept. Microbiology, Milton S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA 17033) Geder, L.; St. Jeor, S.; Michelson-Fiske, S.; Haguenu, F. *J Natl Cancer Inst* 59(1): 55-60; 1977.

The possibility that a herpesvirus (K9V) isolated from a patient with Kaposi's sarcoma was a cytomegalovirus (CMV) was investigated. The host range of K9V, as determined by the induction of virus-specific cytopathology, antigen synthesis, and plaque formation, was limited to human cells and, particularly, to fibroblasts (HEL and WI-38). Immunofluorescence and complement fixation assays confirmed the specificity of the presence of CMV-type antigens in K9V-infected human fibroblasts. In addition, the density of K9V DNA was consistent with the density of CMV DNA. However, the K9V strain of CMV had some peculiarities. The virus seemed more cell-associated in human fibroblasts than known laboratory strains, the spread of the cytopathic effects was slow and did not always involve the whole cell sheet, and total regression of the cytopathic effects was common. Similar characteristics have been observed in the Mj strain of CMV, which is oncogenic in human fibroblasts. (12 refs.)



- 77-3297 **Human Papilloma Viruses.** (Ger.) zur Hausen, H. (Institut für Klinische Virologie, Universität Erlangen-Nürnberg, Loschgestrasse 7, 8250 Erlangen, W. Germany) *Arzneim Forsch* 27(1b): 212-215; 1977.

Clinical and epidemiological differences were established for Verrucae vulgares, plantares, planae, and seborrheicae, as well as genital warts (Condylomata acuminata) and laryngeal papillomas. Biochemical and serological tests established two distinct groups of human wart virus types, herpes papilloma virus (HPV)-1-3 and HPV-40. A possible correlation between condylomas and genital carcinomas is discussed. (10 refs.)

- 77-3298 **Human Cells Transformed In Vitro by Human Cytomegalovirus: Tumorigenicity in Athymic Nude Mice.** (Eng.) Geder, L. (Dept. Microbiology, Milton S. Hershey Medical Center, Pennsylvania State Univ., Coll. Medicine, Hershey, PA 17033) Kreider, J.; Rapp, F. *J Natl Cancer Inst* 58(4): 1003-1009; 1977.

Fifty-three athymic nude mice were inoculated sc with human embryo lung cells transformed in vitro by human cytomegalovirus (CMV). Of the inoculated animals, 62% developed tumors after an av latent period of 19 days. The tumors were composed of small, polygonal cells with large nuclei and a scant cytoplasm embedded in an abundant collagenous matrix. The cells were poorly differentiated and may have been of epithelial origin. Adjacent structures were rarely invaded. CMV-related intracellular and membrane antigens were detected by indirect and anticomplement immunofluorescence techniques in cells cultured in vitro from the tumors. The results provide further evidence of the oncogenic potential of CMV in its natural host. (13 refs.)

- 77-3299 **Oncogenicity of a Nude Mouse Cell Line Transformed by a Human Papovavirus.** (Eng.) Costa, J. (Lab. Pathology, Div. Cancer Biology and Diagnosis, NCI, NIH, Public Health Service, U. S. Dept. Health, Education, and Welfare, Bethesda, MD 20014) Howley, P. M.; Legallais, F.; Yee, C.; Young, N.; Rabson, A. S. *J Natl Cancer Inst* 58(4): 1147-1149; 1977.

Primary cultures of NIH nude mouse (nu/nu) kidney cells were transformed with a human papovavirus (MMV) isolated from a brain tumor of a patient with the Wiskott-Aldrich syndrome. The transformed cell line expressed T antigen, and MMV DNA was found to be associated with the cell DNA. When NIH nu/nu mice were inoculated with the transformed cells, they developed tumors at the injection site but failed to generate detectable levels of T antibody. Control nu/+ littermates rejected the tumor inoculum but mounted an antibody response to T antigen. Nude mouse cells may be a suitable system to test the oncogenicity of in vitro transformed cells. (10 refs.)

- 77-3300 **Search for Oncogenic Viruses in Human Prostate Cancer.** (Eng.) Dmochowski, L. (Dept. Molecular Carcinogenesis and Virology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., 6723 Bertner Ave., Houston, TX 77030) Ohtsuki, Y.; Seman, G.; Maruyama, K.; Knesek, J. E.; East, J. L.; Bowen, J. M.; Yoshida, H.; Johnson, D. E. *Cancer Treat Rep* 61(2): 119-127; 1977.

Ultrastructural, immunological, and biochemical studies were conducted on various types of prostatic tissues. Upon electron microscopic examination, intracisternal viruslike particles were observed in 7/37 human prostatic carcinoma (PCa) specimens. In addition, C-virus particles were observed in 5/37 cases of PCa and in 1/9 cases of benign prostatic hyperplasia (BPH). In normal prostatic tissue from old mice (8 mo-2 yr) of high-mammary-cancer strains, B-type virus particles were found. Sera of PCa (38%) and BPH (25%) patients as well as some normal donors (27%) gave positive cytoplasmic reactions in fixed immunofluorescence (FIF) tests using mouse prostatic cells infected with Soehner-Dmochowski murine sarcoma virus (SD-MSV). Eleven sera positive by FIF produced ferritin labeling of C-type virus particles in SD-MSV-infected cells. Data from molecular hybridization studies indicate that human prostate tissues share very few nucleotide sequences with MSV-M (Moloney strain) and MSV-Ki (Kirsten strain). Cellular RNA's from some PCa and BPH tissues hybridized to higher levels with viral DNA probes than cellular RNA's from normal prostatic tissue. (27 refs.)

- 77-3301 **Human Leukemic Leukocytes: Direct In Vitro Labeling in Short Term Tissue Culture of an Intracytoplasmic RNA with Properties of Known RNA Tumor Viruses (Meeting Abstract).** (Eng.) Meyskens, F. L. (Lab. Tumor Cell Biology, NCI, Bethesda, MD) Gillespie, D. H.; Gallo, R. C.; Saxinger, W. C. *Proc Am Assoc Cancer Res* 18: 82; 1977. (no refs.)

- 77-3302 **Serological Analysis of Cellular and Viral DNA Polymerases by an Antiserum to DNA Polymerase  $\gamma$  of Human Lymphoblasts.** (Eng.) Robert-Guroff, M. (Lab. Tumor Cell Biology, NCI, NIH, Bethesda, MD 20014) Gallo, R. C. *Biochemistry* 16(13): 2874-2880; 1977.

The preparation of an antiserum to highly purified DNA polymerase  $\gamma$  of human lymphoblasts (NC37 cells) and its initial use in the serological analysis of cellular and viral DNA polymerases are reported. The antiserum does not possess enzyme neutralizing activity, but it does bind specifically to DNA polymerase  $\gamma$ . When tested in a double-antibody immunoprecipitation assay, the antibody does not cross-react with DNA polymerase  $\alpha$  or  $\beta$  (purified from NC37 cells) or

with the reverse transcriptase of avian, murine, or primate RNA tumor viruses. Antisera prepared against purified reverse transcriptases similarly do not recognize DNA polymerase  $\gamma$ , either in an enzyme neutralization assay or in the more sensitive double-antibody immunoprecipitation assay. The availability of an antiserum to DNA polymerase  $\gamma$  will allow the further characterization of enzyme activities isolated from cellular material and suspected of being related to viral reverse transcriptases. In those cases in which these activities do not immunologically resemble known viral DNA polymerases, the anti-DNA polymerase  $\gamma$  will help determine the viral or cellular nature of the unknown activity. (38 refs.)

**77-3303 DNA Polymerase  $\gamma$  of Human Lymphoblasts.** (Eng.) Robert-Guroff, M. (Lab. Tumor Cell Biology, NCI, NIH, Bethesda, MD 20014) Schrecker, A. W.; Brinkman, B. J.; Gallo, R. C. *Biochemistry* 16(13): 2866-2873; 1977.

DNA polymerase  $\gamma$ , a component of normal cells, exhibits several properties similar to those of the DNA polymerase of RNA tumor viruses (reverse transcriptase). A highly purified (9,000-fold) DNA polymerase  $\gamma$  from a human lymphoblast cell line (NC37) was examined for its distinguishing features with regard to reverse transcriptase and its possible relationship to the viral enzyme. The final enzyme preparation was demonstrably free of other DNA polymerase activities by immunological and biochemical criteria. Only one form of the enzyme was detected, and it has a molecular wt of 120,000. The enzyme is moderately sensitive to N-ethylmaleimide, exhibits a broad pH optimum around 7.4 in imidazole buffer, and is stimulated by ammonium sulfate. Like reverse transcriptase, DNA polymerase  $\gamma$  prefers (dT)<sub>12-18</sub>(A)<sub>n</sub> to (dT)<sub>12-18</sub>(dA)<sub>n</sub> as a template with either Mg<sup>2+</sup> or Mn<sup>2+</sup> present. Unlike the viral enzyme, its activity with (dG)<sub>12-18</sub>(C)<sub>n</sub> is low, and it does not transcribe the heteropolymeric portions of natural RNA templates. The NC37 enzyme did not copy (dG)<sub>12-18</sub>poly(2'-O-methylcytidylate), confirming previous transcriptase. However, the reverse transcriptase of avian, murine, and primate RNA tumor viruses did not use the template with great efficiency. These reverse transcriptases do not transcribe RNA-primed DNA templates; in contrast, DNA polymerase  $\gamma$  exhibits good activity with these templates. The problems of distinguishing reverse transcriptase and DNA polymerase  $\gamma$  in cells are discussed with respect to these properties. (35 refs.)

**77-3304 Antigenic Relatedness of the DNA Polymerase of Human Breast Cancer Particles to the Enzyme of the Mason-Pfizer Monkey Virus.** (Eng.) Ohno, T. (Inst. Cancer Res., Coll. Physicians and Surgeons, Columbia

Univ., 701 W. 168th St., New York, NY 10032) Spiegelman, S. *Proc Natl Acad Sci USA* 74(5): 2144-2148; 1977.

Antibodies produced by immunizing rabbits with purified DNA polymerase, or reverse transcriptase (RT), from Mason-Pfizer monkey virus (MPMV) cross-reacted with RT from human breast cancer particles. These particles, which were isolated from malignant human breast tumors, are 600S in size and consist of an outer membrane surrounding a core of RT complexed to a 70S RNA species. The cross-reactivity was shown by inhibition of enzyme activity and by complex formation between purified enzyme and isolated immunoglobulin against MPMV polymerase. No such interactions were observed with other oncornaviral RT's of avian, murine, feline, or simian origin. Further, the immunoglobulin failed to neutralize the RT's from human mesenchymal neoplasias (leukemias and lymphomas) or the activities of normal cellular DNA polymerases ( $\alpha$ ,  $\beta$ ,  $\gamma$ ). Since MPMV can be produced in tissue culture in yields adequate for the purification of its RT, the use of antisera to the enzyme may be of value as a diagnostic aid in human breast cancer. (25 refs.)

**77-3305 Polyomavirus in Urine in Pernicious Anaemia (Letter to Editor).** (Eng.) Lecatsas, G. (Dept. Microbiology, Univ. Pretoria, Pretoria, South Africa) Pretorius, F.; Crewe-Brown, H.; Requadt, E.; Ackthun, I. *Lancet* 2(8029): 147; 1977.

Polyomaviruses were found in the urine of a 65-yr-old man with pernicious anemia. These potentially oncogenic viruses may replicate in immunodeficient patients, particularly when immunosuppressive therapy is employed. (7 refs.)

**77-3306 In Situ Hybridization Analysis of Polyoma DNA Replication in an Inducible Line of Polyoma-transformed Cells.** (Eng.) Neer, A. (Dept. Biology, Technion-Israel Inst. Technology, Haifa, Israel) Baran, N.; Manor, H. *Cell* 11(1): 65-71; 1977.

Unlike noninducible polyoma-transformed cells whose resident viral genome cannot be activated, inducible polyoma-transformed rat embryo muscle LPT cells contain at least 20 genome equivalents of extrachromosomal viral DNA per cell. To examine the possibility that this DNA is in the form of plasmids, the distribution of viral DNA molecules among individual cells of LPT clone 1a was determined using in situ hybridization and DNA reassociation kinetics. The results demonstrate that the viral DNA is not equally distributed among the cells, as would be expected if all the extrachromosomal viral DNA molecules were plasmids, but that 0.04%-0.25% of the cells in each culture are spontaneously induced to synthesize about 24,000 genome equivalents of



viral DNA per cell. This type of distribution is expected if the cells contain intracellular DNA that is excised and replicated autonomously only after induction. It was also found that: (1) in cultures exposed to mitomycin C, the percentage of induced cells remains low (as in untreated cultures) for about 9 hr and then increases to 30%-57% as more and more cells are asynchronously recruited to replicate the viral DNA; (2) all the viral DNA molecules were found within nuclei, and many were clustered in aggregates containing up to 2,000 genome equivalents. (14 refs.)

- 77-3307 Isolation of Variant Cells with Defective Metabolic Cooperation (MEC-) from Polyoma Virus Transformed Syrian Hamster Cells.** (Eng.) Wright, E. D. (Royal Free Hosp., Sch. Medicine, 8 Hunter St., London WC1N 1BP, England) Goldfarb, P. S.; Subak-Sharpe, J. H. *Exp Cell Res* 103(1): 63-77; 1976.

Two selection systems designed to obtain metabolic cooperation negative (mec-) cells from a parental metabolic cooperation positive (mec+) population are described. Variant cell lines derived from polyoma virus transformed BHKC13 cell line PyY were used. In both systems,  $9 \times 10^5$  donor cells were mixed in suspension with  $1 \times 10^5$  recipients, seeded, and allowed to spread. In system I, 1 mg/ml 5-bromodeoxyuridine (BUdR) was added, and in system II, 100  $\mu$ g/ml 8-azaadenine (8-AA) was added. The cultures were then incubated for 2-3 days. The selection I cocultures were exposed to blue light for 30 min. The medium was replaced with fresh medium plus analogue, and the cultures were again incubated for 2-3 days. Surviving cells were removed and  $1 \times 10^5$  of these were mixed with  $9 \times 10^5$  fresh donor cells. This was repeated for each subsequent round of selection. In these systems, cells resistant to pyrimidine deoxyribonucleoside analogues or purine analogues were used as recipients, and cells sensitive to these analogues were used as donors. When cooperation occurs, donor cells take up the analogue and transfer nucleotide derivatives to the recipients, resulting in the death of both the donor and cooperating recipient. The recipients that do not cooperate survive. The mec- cells are smaller and more epithelioid than the mec+ cells. The mec- cells contained only half the chromosome number of the mec+ parental cells. The 8-AA system did not produce cells with reduced ability for metabolic cooperation. (22 refs.)

- 77-3308 Simian Virus 40 Facilitates Multiplication of Replication Defective Mutants of Polyoma Virus in BALB/3T3 Mouse Cells.** (Eng.) Hakura, A. (Dept. Tumor Viruses, Res. Inst. Microbial Diseases, Osaka Univ., Suita, Osaka, Japan) *Nature* 267(5611): 528-529; 1977.

Two host range mutants of polyoma virus, HR-86 and HR-101, were isolated to examine the relationship between the

viral gene functions of polyoma virus and simian virus 40 (SV40). When A31-714 cells (subclone of mouse BALB/3T3) and SA31 cells (SV40-transformed A31-714 cells) were infected with HR-101, its plaque-forming ability and its ability to multiply were reduced on the A31-714 cells. Coinfection of A31-714 cells with HR-101 [multiplicity of infection (MOI), 0.03] and SV40 (MOI, 60) enhanced the growth of HR-101 approx 60-fold. Early events of SV40 infection appear to convert the susceptibility of mouse cells to infection with these mutants from a restrictive to a nonrestrictive state. The defect in virus growth of the mutants may be helped either by the direct action of SV40 gene products or by an indirect action in which a SV40 genome in these cells alters the expression of cellular genes. (13 refs.)

- 77-3309 Suppression of Replication of SV40 and Polyoma Virus in Mouse-Human Hybrids.** (Eng.) Huebner, K. (Wistar Inst. Anatomy and Biology, 36th St. at Spruce, Philadelphia, PA 19104) Shander, M.; Croce, C. M. *Cell* 11(1): 25-33; 1977.

Murine and human cells, permissive respectively for polyoma virus and simian virus 40 (SV40) replication, were fused, and the resulting heterokaryons and hybrids were tested with virus for permissiveness. The murine cell lines were Balb/c MPM, C57BL MPM, and OTT 6050 (solid teratocarcinoma); the human cell line was HT 1080 (fibrosarcoma). All the heterokaryons produced were permissive for both types of virus, but hybrids that segregated human chromosomes were permissive only for polyoma virus and hybrids that segregated murine chromosomes were permissive only for SV40. This indicates that during the transition from heterokaryon to hybrid cell, suppression of expression of species-specific functions, such as those required for the replication of these species-specific viruses, occurs in parallel with the direction of chromosome loss and suppression of nucleolus organizer activity. (42 refs.)

- 77-3310 Release and Characterization of a Growth Factor for SV40 Virus-Transformed Cells from Human Platelets (Meeting Abstract).** (Eng.) Kohler, N. (Milton S. Hershey Medical Center, Pennsylvania State Univ., Hershey, PA 17033) Lipton, A. *Proc Am Assoc Cancer Res* 18: 244; 1977. (no refs.)

- 77-3311 A Cell Membrane Alteration Specifically Induced by SV40 Transformation.** (Eng.) Lago, C. (International Inst. Genetics and Biophysics, C.N.R., Via

Marconi 10, 80125, Italy) Sartorius, B.; Tramontano, D.; Amati, P. *J Cell Physiol* 92(2): 265-274; 1977.

Transformation of mouse 3T3 cells by simian virus 40 (SV40) resulted in a specific membrane modification that rendered the cells resistant to the killing action of the lipophilic antibiotic amphotericin B. This alteration is under genetic cellular control, as transformed cell variants (flat revertants) also revert to sensitivity to the antibiotic. The alteration is specific for SV40 transformation, since transformation with polyoma or mouse sarcoma viruses does not confer resistance to amphotericin B. Analogous resistance is induced by SV40 transformation of primary human fibroblast cells. The acquired resistance is not due to decreased binding of amphotericin B and is partially reversed if cells are grown in the presence of cholesterol. It is suggested that SV40 transformation involves a specific change to the sterol structure of the membrane or the loss of a minor cholesterol fraction responsible for the killing action of the antibiotic. (24 refs.)

**77-3312 Cell-Density-dependent Increase in Chromatin-associated ADP-Ribosyltransferase Activity in Simian Virus 40-transformed Cells.** (Eng.) Miwa, M. (Dept. Molecular Oncology, Inst. Medical Science, Post Office Takanawa, Tokyo 108, Japan) Oda, K.; Segawa, K.; Tanaka, M.; Irie, S.; Yamaguchi, N.; Kuchino, T.; Shiroki, K.; Shimojo, H.; Sakura, H.; Matushima, T.; Sugimura, T. *Arch Biochem Biophys* 181(1): 313-321; 1977.

The ADP-ribosyltransferase (ADP-RT) activities of a variety of simian virus 40 (SV40)-transformed and nontransformed cells were compared under various growth conditions. Among cells harvested at confluency, the ADP-RT activity associated with chromatin was 2-10 times higher in the transformed cells [with a range of 9.2-40.4 nanomoles (nmol) of ADP incorporated/mg protein] than in the nontransformed cells (0.8-4.7 nmol/mg). When confluent transformed cells were subcultured, their specific ADP-RT activity first decreased two- to fourfold and then rapidly increased during the logarithmic growth phase. This increase ceased or slowed down when the cells entered the stationary phase. In contrast, the activity of the nontransformed cells remained low throughout the growth cycle. In temperature-sensitive (ts) SV40ts-transformed cells, this density-dependent increase in ADP-RT activity was seen at the permissive temperature, but the activity remained low during growth at a restrictive temperature. The higher level of ADP-RT activity in transformed cells probably results from an increase in the amount of enzyme or acceptors associated with chromatin rather than from a steric alteration of the enzyme molecules in chromatin. The nonhistone proteins in transformed and nontransformed cells may differ both qualitatively and quantitatively. The increased modification of chromosomal proteins in transformed cells may alter their interaction with DNA and result in altered patterns of gene expression and growth. (no refs.)

**77-3313 African Green Monkey Fibroblast Actin Morphology During SV40 Infection.** (Eng.) Brandner, G. (Institut für Virologie der Universität Freiburg, Postfach 820, D-7800 Freiburg, W. Germany) Cho, M. S. *Z Naturforsch (C)* 32(5/6): 409-412; 1977.

Monkey skin fibroblasts were infected with 0.5 to  $1 \times 10^8$  median infective tissue culture doses/ml of simian virus 40 (SV40) strains 777 or ELO. Cells that exhibited the viral tumor antigen retained the normal morphology of actin filaments up to 6 days after infection with both SV40 strains. However, when the cells were transformed (using SV40 ELO; 10-50 foci of transformed cells/culture after 2-4 wk), they lost the normal actin morphology. (13 refs.)

**77-3314 Inhibition of Simian Virus 40 Replication by Interferon Treatment Late in the Lytic Cycle.** (Eng.) Jakobson, E. (Dept. Virology, Weizmann Inst. Science, Rehovot, Israel) Revel, M.; Winocour, E. *Virology* 80(1): 225-228; 1977.

BAC-1 or CV-1 monkey cells were exposed to homologous interferon (IF) (100 U/ml) before and at different times after infection with simian virus 40 (SV40, 25 plaque-forming units/cell). Exposure of infected cells to IF after the onset of viral DNA synthesis inhibited subsequent rounds of viral DNA replication and overall virus yields as effectively as did IF treatment prior to infection. In this respect, the lytic SV40-monkey cell system differs from other lytic virus-cell systems in which the antiviral state develops maximally only when IF is applied prior to infection. (13 refs.)

**77-3315 Inhibition of Simian Virus 40 DNA Synthesis in Yaba Virus-preinfected Cells.** (Eng.) Taylor, J. L. (Lab. Molecular and Cancer Virology, Dept. Microbiology, Southern Illinois Univ., Carbondale, IL 62901) Rouhandeh, H. *Biochim Biophys Acta* 475(2): 276-280; 1977.

The effect of Yaba virus preinfection on DNA synthesis in simian virus 40 (SV40)-infected Jinet cells was evaluated by time-course synthesis studies using the incorporation of labeled thymidine. Yaba virus preinfection (3 focus-forming units/cell) inhibited SV40 DNA synthesis when the elapsed time between Yaba virus and SV40 infections (1.5 plaque-forming units/cell) was 3 days. This inhibition was demonstrated by hybridization studies and sedimentation analysis. In addition, the usual stimulation of cellular DNA synthesis induced by SV40 infection was inhibited. This inhibition occurred at a time late in Yaba virus infection when no cytoplasmic Yaba virus-specific DNA synthesis occurred. The exact mechanism of inhibition of SV40 virus by Yaba virus was not determined. (17 refs.)



- 77-3316 Episomal Viral DNA in a *Herpesvirus saimiri*-transformed Lymphoid Cell Line. (Eng.) Werner, F. J. (Institut für Klinische Virologie der Universität, Erlangen-Nürnberg, 8520 Erlangen, W. Germany) Bornkamm, G. W.; Fleckenstein, B. J. *Virology* 22(3): 794-803; 1977.

The isolation of nonintegrated, circular, *Herpesvirus saimiri* DNA molecules from a transformed lymphoid cell line and the structural analysis of this DNA by partial denaturation are described. Closed circular, superhelical, viral DNA molecules were isolated using isopycnic centrifugation in cesium chloride, followed by sedimentation through glycerol gradients and equilibrium centrifugation in cesium chloride-ethidium bromide. The isolated circular molecules had an average molecular weight of  $131.5 \times 10^6$ . Partial denaturation revealed molecules differing remarkably in degree of denaturation. Of a total of 65 molecules observed, 46 did not exhibit any single-stranded loops; 19 molecules showed denatured regions comprising 22.4% to 53.8% of the total length. The short light (L) regions had contour lengths corresponding to a molecular weight of  $31.5 \times 10^6$ ; the long L regions corresponded to a molecular weight of  $54.0 \times 10^6$ . The lengths of the short and long heavy (H) regions in the circles corresponded to molecular weights of  $20.0 \times 10^6$  and  $25.6 \times 10^6$ , respectively. All long L regions except one were aligned in the same polarity, revealing two distinct areas containing preferentially native DNA and three areas containing preferentially single-stranded DNA. Maps of the L regions indicated that the short L regions were subsets of the long L regions. Sequences of both L regions had the same orientation. Circular molecules from *H. saimiri*-transformed lymphoid cells of line 1670 appeared to represent defective genomes that contained only 75% of the genetic information in the L-DNA of *H. saimiri* virions. (19 refs.)

- 77-3317 Electron Microscopic Study of *Herpesvirus Saimiri*. (Eng.) Tralka, T. S. (Lab. Pathology, NCI, Bethesda, MD 20014) Costa, J.; Rabson, A. *Virology* 80(1): 158-165; 1977.

The replication of *herpesvirus saimiri* ( $10^4$  plaque-forming units/ml) was studied by electron microscopy in highly permissive primary owl monkey kidney cells (OMK) and less permissive Vero African green monkey kidney cells. In OMK cells, toroid structures were observed in the nucleoid of immature and mature virions, and what has been described as intranuclear envelopment was shown to be envelopment by budding into intranuclear vesicles. The membranes of the vesicles were continuous with the inner nuclear membrane. In the less permissive Vero cells, accumulation of empty cytoplasmic capsids associated with dense osmiophilic material suggested the possibility of cytoplasmic assembly of defective particles. Other unusual structures associated with virus replication in the Vero cells are described, and their possible

role in virus morphogenesis is discussed. The structures included tubular lamella-particle complexes and fibrils that may be virus DNA. (19 refs.)

- 77-3318 Biochemical and Immunological Properties of Squirrel Monkey Retrovirus: A New Virus Isolated from a New World Monkey (Meeting Abstract). (Eng.) Schochetmen, G. (Frederick Cancer Res. Center, Frederick, MD 21701) Fine, D. L.; Heberling, R. L. *Proc Am Assoc Cancer Res* 18: 110; 1977. (no refs.)

- 77-3319 Biologic and Biochemical Characterization of a New Isolate of Endogenous C Type Virus of a *Papio papio* Baboon. (Fre.) Rhodes-Feuillette, A. (Laboratoire d'Hématologie expérimentale, U.E.R. d'Hématologie, Institut de Recherches sur les Leucémies, hôpital Saint-Louis, 75010 Paris, France) Ravicovitch, R. E.; Lasneret, J.; Canivet, M.; Peries, J. *CR Acad Sci (D) (Paris)* 284(21): 2183-2185; 1977.

A new strain of endogenous C-type virus was isolated from the testis of a *Papio papio* baboon. The strain, designated B32 virus, was characterized biochemically and biologically. Its host range differs from that of the prototype strain of the same virus. B32 virus replicates in rhesus monkey, human dog, mink, and hairless mouse cells, but not in normal mouse and rat cells. (6 refs.)

- 77-3320 Model Studies of Virus-induced Tumors and Their Immunological Treatment. (Ger.) Schafer, W. (Max-Planck-Institut für Virusforschung, Tübingen, W. Germany) *Naturwiss Rundsch* 30(4): 133-136; 1977.

The sources and general biological properties of oncornaviruses, in particular the structure and serobiological properties of the C-type viruses and surface antigens of host cells, are discussed. Pure glycoprotein gp71 had an active immunizing effect on mice infected with exogenous Friend leukemia virus, but it had no effect on endogenous virus. In passive immunization (serum therapy) studies, gp71 antibodies produced in rabbits and goats were effective against leukemia viruses in mice and cats and against a sarcoma virus in cats. This indicates that it is theoretically possible to produce a therapeutic effect in virus-induced leukemias and sarcomas solely with virus-specific antibodies and that these antibodies are also effective in C-type virus infections of a

different mammalian species. The application of an immunological method to humans is mentioned. (no refs.)

**77-3322 A Plasminogen Activator Correlated with Oncogenic Transformation (Meeting Abstract).** (Eng.) Unkeless, J. C. (Rockefeller Univ., New York, NY) *Diss Abstr Int [B]* 37(8): 3730; 1977. (no refs.)

**77-3321 Rapid Oncornavirus Detection Using Critical Point Drying, Biochemical and Density Scans (Meeting Abstract).** (Eng.) Erlick, B. J. (Cancer Inst., Hahnemann Medical Coll., Philadelphia, PA 19102) West, W. W.; Fuscaldo, K. E. *Proc Am Assoc Cancer Res* 18: 96; 1977. (no refs.)

\* (Rev): 77-3026, 77-3027, 77-3028, 77-3029, 77-3030, 77-3033, 77-3037.

\* (Chem): 77-3122, 77-3152, 77-3200, 77-3204.

\* (Immun): 77-3325, 77-3326, 77-3341.

\* (Path): 77-3352, 77-3368, 77-3375, 77-3394, 77-3462, 77-3464, 77-3483, 77-3491.



- 77-3323 **Passive Local Anaphylaxis: Demonstration of Antitumor Activity and Complementation of Intratumor BCG.** (Eng.) Lynch, N. R. (Laboratoire d'Immunopathologie, Institut de Recherches Scientifiques sur le Cancer, B.P. No. 8, 94800 Villejuif, France) Salomon, J. C. *J Natl Cancer Inst* 58(4): 1093-1098; 1977.

When an extracellular dye, Lissamine green, or  $^{51}\text{Cr}$ -labeled spleen cells were injected iv into C3H mice bearing small, partially necrotic 3-methylcholanthrene-induced transplantable fibrosarcomas (McC3), the tumor content of these circulating elements per unit wt was substantially lower than that of other selected organs. The level of these bloodborne materials was, however, significantly augmented by the intratumor induction of passive local anaphylaxis (PLA). The PLA-induced augmentation was inhibited by administration of the histamine and serotonin antagonist cyproheptadine. Comparable increases were also induced by the intratumor injection of a histamine and serotonin mixture or BCG. The weekly intratumor induction of PLA in McC3 tumors resulted in complete regression of a significant number of the tumors; this therapeutic effect was eliminated by cyproheptadine treatment. The intratumor injection of BCG induced the regression of approx 50% of injected tumors, and the combination of this immunostimulant treatment with the generation of PLA was more therapeutically effective than either treatment alone. PLA in the vicinity of solid tumors may, by increasing vascular permeability, potentiate antitumor effector mechanisms, particularly when these are BCG-stimulated. Despite this demonstration of a possible role of anaphylactic reactions in tumor immunity, no definitive evidence was found that active reagin-mediated local anaphylaxis occurs in C3H mice bearing the McC3 tumor, whether or not they were treated with immunostimulants. (26 refs.)

- 77-3324 **Relationship of Host Immune Status to Tumor Cell Arrest, Distribution, and Survival in Experimental Metastasis.** (Eng.) Fidler, I. J. (Basic Res. Program, Frederick Cancer Res. Center, Post Office Box B, Frederick, MD 21701) Gersten, D. M.; Riggs, C. W. *Cancer* 40(1): 46-55; 1977.

The kinetics of initial arrest in organs, distribution, survival, and fate of  $^{125}\text{I}$ -iododeoxyuridine-labeled B16 melanoma tumor cells injected iv into normal, tumor-sensitized, and immune manipulated syngeneic (C57BL/6) and allogeneic (A) mice were investigated. Groups of animals were killed at intervals ranging from 2 min to 14 days after tumor cell injection.

Lungs, liver, spleen, and blood were collected from each animal and processed so that the radioactivity associated with DNA of tumor cells viable at the time of sacrifice could be monitored. Although initial tumor cell arrest in organs was influenced by the host immune status, it did not correlate with survival kinetics or development into tumors. The same tumor that was rejected in mice after sc tumor challenge grew in the lungs after iv injection. Therefore, rejection of sc challenge as the sole criterion of host immunity to neoplasms should be questioned. Allogeneic animals are not appropriate model systems for the study of experimental metastasis. Animals sensitized to a tumor exhibited kinetic patterns of tumor cell arrest and survival that differ from those of normal syngeneic hosts. (27 refs.)

- 77-3325 **Effect of Immune Manipulation on Natural Immune Responses to Murine Mammary Tumor Antigens.** (Eng.) Blair, P. B. (Dept. Bacteriology and Immunology, Univ. California, Berkeley, CA 94720) Lane, M. A. *J Natl Cancer Inst* 59(1): 251-257; 1977.

The virus-host relationship in BALB/cfC3H virgin female mice neonatally infected with murine mammary tumor virus (MuMTV) was significantly altered by brief immunosuppressive treatment with antithymocyte globulin (ATG: 11 ip injections, each containing 0.5-ml equivalents of antiserum) during young adult life. Natural immune responses were augmented and changed. The ability of lymphoid cells to attack target cells expressing MuMTV-associated antigens was significantly increased, and a new component, non-T-cell reactivity, was added to the T-cell reactivity found in normal females. Serum blocking factors were quantitatively and qualitatively altered. The sera from a significant number of ATG-treated females did not effectively protect pretreated target cells against attack by their own spleen cells. (15 refs.)

- 77-3326 **Type C Virus Expression and Host Response in Diet-cured NZB/W Mice.** (Eng.) Gardner, M. B. (Dept. Pathology, Univ. Southern California, Sch. Medicine, Los Angeles, CA 90033) Ihle, J. N.; Pillarisetty, R. J.; Talal, N. *Nature* 268(5618): 341-344; 1977.

The effect of a low protein diet in NZB/W mice was investigated to determine whether the resulting freedom from autoimmune disease was due to a decrease in the production

of endogenous MuLV or a reduction in the host response to the virus. Disease prevention was not due to either of these events. (29 refs.)

77-3327 **Study of Host Immunological Resistance to Syngeneic Lymphoma Using Whole Body Irradiation.** (Eng.) Maruyama, Y. (Dept. Radiation Medicine, Univ. Kentucky A. B. Chandler Medical Center, Lexington, KY 40506) *Int J Radiat Oncol Biol Phys* 1(11/12): 1159-1169; 1976.

Whole body irradiation was studied to determine its value in detecting minimal degrees of immunogenicity and host-resistance against a syngeneic, weakly antigenic tumor. Inbred C57BL mice were irradiated with dose of 375 rads delivered from several hr to about 1 day before challenge with tumor cells. Heavily irradiated (HI) cells were prepared by irradiating cells to 9,000 rads in vitro. The tumor system employed was the LSA ascites lymphoma. When nonirradiated cells were used, the tumor dose for 50% (TD<sub>50</sub>) values obtained were not affected by the prior irradiation of the recipients or by dead cells. The addition of x-ray-killed cells also failed to alter TD<sub>50</sub> values. When cells irradiated with doses  $\geq 1,125$  rads were injected into nonirradiated and preirradiated recipients, discernible differences became evident. The radiation-damaged cell was more readily recognized by the immune competent host. In sham handled mice,  $3.0 \times 10^5$  cells treated with 1,125 rads produced 50% tumor takes. In irradiated recipients, the LD<sub>50</sub> (leukemic cell dose/50% takes) was reduced to  $6.8 \times 10^4$  cells. When 1,975-rad x-ray-treated cells were used, the irradiated mice required a LD<sub>50</sub> cell dose of  $2.6 \times 10^7$  cells, and the unirradiated mice, a LD<sub>50</sub> dose of  $2.6 \times 10^8$  cells. Significant differences were observed when the same number of irradiated cells were injected into the irradiated or unirradiated mice. Only 9 tumor takes were detected in the 40 unirradiated mice inoculated, whereas 29 takes were found among the 40 irradiated mice. The results of serial sublethal viable tumor challenge using small numbers of tumor cells showed that the number of surviving animals declined, but in the survivors, tumor resistance increased. A subsequent million-fold increase in host-resistance against tumor challenge was achieved. The use of serial exposures to viable tumor was successful in producing immunity to tumor challenge. Whole body irradiation was a sensitive method for the detection of minimally antigenic properties of the LSA lymphoma in syngeneic C57BL mice. (34 refs.)

77-3328 **Immunologic Aspects of Ultraviolet Carcinogenesis (Meeting Abstract).** (Eng.) Fisher, M. S. (Univ. Utah, Salt Lake City, UT 84112) *Diss Abstr Int [B]* 38(1): 135; 1977. (no refs.)

77-3329 **Pulmonary Metastases, a Potential Biologic Consequence of Anesthetic-induced Immunosuppression by Thiopental.** (Eng.) Lundy, J. (Dept. Surgery, Univ. Connecticut Health Center, Farmington, CT 06032) Lovett, E. J.; Conran, P. *Surgery* 82(2): 254-256; 1977.

The effects of the anesthetic agent thiopental on the development of pulmonary metastases were examined in female C57Bl/6 mice injected iv with  $5 \times 10^4$  cells of a syngeneic, 20-methylcholanthrene-induced fibrosarcoma. In two separate experiments, mice treated with thiopental (37.5 mg/kg, ip) had a significantly higher incidence of pulmonary metastases than control animals (45 vs 25-27 metastases). This correlated with an impaired delayed hypersensitivity reaction to the de novo antigen 2,4-dinitrochlorobenzene (DNCB) and a suppressed mixed leukocyte culture (MLC) reaction. However, if animals were sensitized with DNCB 5 days prior to receiving thiopental, DNCB reactivity was not impaired. This suggests strongly that the observed defect is in the afferent arm of the immune response. Thiopental suppresses cell-mediated immune responses in this system, and the observed biologic consequence is an increase in pulmonary metastases. (12 refs.)

77-3330 **Defective Recognitive Immunity in Family Aggregates of Colon Carcinoma.** (Eng.) Berliner, N. T. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021) Lopez, C.; Lipkin, M.; Vogel, J. E.; Good, R. A. *J Clin Invest* 59: 761-769; 1977.

In vitro measurements were made of the mixed leukocyte culture (MLC) responsiveness of 5 patients with adenocarcinoma of the colon and 18 unaffected blood relatives. Three of the five affected members demonstrated a significantly decreased MLC responsiveness ( $< 68\%$  relative response). Eight of the 18 unaffected individuals (44%) also showed decreased cell-mediated immune capacity, as measured by MLC responsiveness of the FH cells (Ficoll-Hypaque-isolated mononuclear cells). Seven of these eight and 2/3 affected members could produce normal responses when cells isolated by G-10 column filtration (to eliminate adherent cells) were used. A generalized hyporesponsiveness to allogeneic stimuli similar to that observed in cancer patients was found in all unaffected individuals with deficient MLC responses. Evidence suggests that deficiencies of recognitive immunity appear to aggregate in families in which colon carcinoma aggregates. A genetically transmitted immunologic determinant may contribute to the genesis of malignancy in some of these families. (39 refs.)

77-3331 **In Vitro Biosynthesis of Cellular ABO Antigens: Defective Expression on Neoplastic Cells**



(Meeting Abstract). (Eng.) Kuhns, W. J. (New York Univ. Sch. Medicine, New York, NY) *Am J Pathol* 86(2): 69a; 1977. (no refs.)

- 77-3332 **HLA Linked Resistance Factors and Survival in Acute Myelogenous Leukemia.** (Eng.) Oliver, R. T. (I.C.R.F. Dept. Medical Oncology, St. Bartholomew's Hosp., London EC1, England) Pillai, A.; Klouda, P. T.; Lawler, S. D. *Cancer* 39(6): 2337-2341; 1977.

The concept that HLA-linked immune response genes may influence the survival of patients with malignant disease was investigated by performing HLA typing on 150 patients with acute myelogenous leukemia. Patients with A1 + B8 and/or A2 + B12 survived longer than patients without either of these pairs of antigens. However, after correction of the statistical probability for the number of A- and B-locus combinations theoretically possible, these differences were not statistically significant. Clarification of the relationship between HLA antigens and survival depends on independent analysis of other series and more direct approaches to the study of immune response genes in man. (22 refs.)

- 77-3333 **HLA Polymorphism and Its Relation to Cervical Neoplasia.** (Ger.) Koenig, U. D. (Institut für Experimentelle Haematologie und Bluttransfusionswesen der Universität Bonn, 53 Bonn-Venusburg, W. Germany) Muller, N. *Fortschr Med* 95(9): 565-568; 1977.

Twenty-three histocompatibility (HLA) antigens were investigated in 121 patients with clinical cervical cancer as well as 188 male and 212 female controls. The patients showed significant differences in some HLA frequencies compared with the controls. In patients with the localized form of the neoplasia, HLA-A3 was decreased and A0 and B12 were increased. In those with the disseminated form, A1 was decreased and AW32 and B12 increased. HLA antigens and other unknown factors, ie immune response genes or herpes simplex virus type 2 infection, could produce an increased susceptibility to the disease. (20 refs.)

- 77-3334 **Possible Effects of Non-HLA Antibodies in Common Typing Sera on HLA Antigen Frequency Data in Leukemia.** (Eng.) Pollack, M. S. (Div. Clinical Immunology, Ortho Diagnostics Res. Inst., Raritan, NJ 08869) Du Bois, D. *Cancer* 39(6): 2348-2354; 1977.

A study of HLA antigen expression in leukemia revealed increased frequencies of HLA-A2 and A3 in patients with acute

lymphocytic leukemia (42), of A9, A28, Aw30, B12, and B18 in patients with chronic lymphocytic leukemia (CLL, 34), and of B14 in patients with acute myelocytic leukemia (26). Selective adsorption of monospecific HLA typing sera with HLA-typed platelets, purified B lymphocytes, cultured lymphoid cells, or lymphocytes from patients with active CLL demonstrated that many of the sera contained antibodies to non-HLA antigens. Antibodies were detected to antigens present on peripheral blood B lymphocytes, cultured lymphoid cells, and leukemic cells from patients with both the myelocytic and lymphocytic forms of leukemia, but they were absent from T lymphocytes and platelets. Since these antibodies are present in a large proportion of common HLA typing sera, caution should be used in interpreting data on HLA antigen associations in leukemia. The non-HLA antibodies may have accounted for some of the increased HLA frequencies observed. (13 refs.)

- 77-3335 **Detection of Thymus Leukemia Antigens on the Surface Membranes of Murine Leukemia Cells Resistant to Thymus Leukemia Antibodies and Guinea Pig Complement.** (Eng.) Liang, W. (La Rabida-Univ. Chicago Inst., Univ. Chicago, E. 65th St. at Lake Michigan, Chicago, IL 60649) Cohen, E. P. *J Natl Cancer Inst* 58(4): 1079-1086; 1977.

The thymus leukemia (TL) antigens of ASL-1 murine leukemia reversibly disappeared from the membranes of cells exposed to TL antisera; the cells acquired resistance to fresh TL antiserum and complement (antigenic modulation). Three independent methods, however, indicated that the acquisition of complement resistance preceded the complete disappearance of TL antigens from the cell surface. Modulated cells reduced known titers of TL antisera by absorption; they stained positively in immunofluorescence studies involving TL antibodies and fluorescence-labeled rabbit anti-mouse immunoglobulin. TL antigens labeled previously with <sup>125</sup>I were recovered by immunoprecipitation from cellular extracts prepared with nonionic detergent. Continued exposure of the cells to TL antiserum led to complete disappearance of the antigens. Similar results were obtained for RADA-1 cells, another murine leukemia that forms TL antigens, although these cells were resistant to the cytolytic effects of TL antisera and guinea pig complement (GPC) without prior exposure to TL antibodies. The density of TL antigens remaining on the surfaces of different TL(+) cell types failed to correlate with resistance to TL antibodies and GPC. Cells from F<sub>1</sub> hybrids of TL(+) and TL(-) mouse strains formed TL antigens and were susceptible to TL antibodies and GPC, even though the density of TL antigens formed by the susceptible cells was less than that of TL antigens formed by modulated cells. Stable somatic hybrids of RADA-1 cells and TL(-) LM cells formed TL antigens at a lower density than did RADA-1 cells, and they lysed in the presence of aliquots of the antisera and GPC used in previous tests. (21 refs.)

- 77-3336 **Tissue-specific Antibodies Against Human Lung and Breast Carcinoma Dehistonized Chromatins.** (Eng.) Chiu, J. F. (Dept. Biochemistry, Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232) Hnilica, L. S.; Chytil, F.; Orrahood, J. T.; Rogers, L. W. *J Natl Cancer Inst* 59(1): 151-154; 1977.

Tissue-specific antisera against human lung and breast carcinoma dehistonized chromatins were prepared in rabbits. The specificity of these antisera was determined by complement fixation. In the presence of antiserum against human lung carcinoma, only chromatins from lung carcinoma fixed complement significantly. Chromatins isolated from human breast carcinoma, HeLa cells, normal lung tissue, breast tissue, or term placenta were negative (ie, inactive). In a similar assay with antiserum against dehistonized breast carcinoma chromatins, only breast carcinoma chromatins fixed complement. Immunohistochemical localization of the antigens by the horseradish peroxidase bridge method demonstrated their presence in the nuclei. The results support the concept that neoplastic disease and its phenotype may reflect altered biochemical control mechanisms and abnormal gene expression. (21 refs.)

- 77-3337 **Alien Histocompatibility Determinants on the Cell Surface of Sarcomas Induced by Methylcholanthrene. II. In Vitro Serological Studies.** (Eng.) Invernizzi, G. (Div. Experimental Oncology A, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy) Carbone, G.; Parmiani, G. *Tumori* 63(2): 181-194; 1977.

An attempt was made to analyze by serological methods the cell surface antigens of two 2-methylcholanthrene-induced BALB/c fibrosarcomas (TZ15 and ST2) that cross-react in vivo with normal allogeneic tissue. In contrast to in vivo transplantation data, no cross-reactions were found between TZ15 and normal AKR cells. Among the cross-reactions observed in vivo between ST2 and 3Hf, DBA/2, and C57BL/6J cells, only normal C57BL/6J antigens were detectable by serology. A new but still undefined system of alien normal antigens shared by C3Hf, AKR, C57BL/6J, NIH lymphoid, and ST2 cells was revealed by BALB/c alloantisera in an isotopic antiglobulin assay. The identification of the alloantigens and their relationship with tumor-associated transplantation antigens require further investigations. (31 refs.)

- 77-3338 **The Heterocytotoxicity of Human Serum. III. Studies of the Serum Levels and Distribution of Activity in Human Populations.** (Eng.) Glumac, G. (Dept. Microbiology and Immunology, Queen's Univ., Kingston,

Ontario, Canada) Mates, A.; Eidinger, D. *Clin Exp Immunol* 26(3): 601-608; 1976.

A metabolic inhibitory (MI) assay was used to measure the levels of serum heterocytotoxicity in normal neonates, normal adults, and cancer patients. The computer analysis of blood samples, separated by age level, did not differ significantly in MI activity. However, in a subgroup of normal adults, it was found that young females taking oral contraceptives exhibited elevated levels in contrast with the non-medicated healthy group. Cancer patients showed elevated levels when compared with all controls. Cancer-bearing males had only slightly elevated levels. Newborns exhibited about half the level of normal adults. A computer analysis showed maximally elevated levels in patients with visceral cancer, particularly in individuals with adenocarcinomas, while patients with sarcomas exhibited reduced levels. Levels in patients undergoing radiotherapy, chemotherapy, or following surgery indicated that therapeutic actions did not substantially influence levels of activity. The data indicate that the metabolic inhibition assay may provide a useful measure of natural antibody and activation of the alternate complement pathway representative of mechanisms of natural immunity. (63 refs.)

- 77-3339 **Immunologic and Electrophoretic Detection of the Chalone-containing Complex in Tissues of Different Origin.** (Rus.) Okulov, V. B. (Lab. Experimental Tumors, N.N. Petrov Res. Inst. Oncology, Acad. Medical Sciences, USSR, Leningrad, USSR) Ketlinski, S. A. *Arkhh Anat Gistol Embriol* (2): 84-88; 1977.

Antiserum was prepared in rabbits against one of the components of a tissue-specific protein complex isolated from the back skin of rats and containing G<sub>1</sub> and G<sub>2</sub> chalones. By means of this antiserum, an antigen identical to the cutaneous antigen was detected in the mucous membrane of the tongue, esophagus, prestomach, and vagina and in the epidermis of the tail and sole. The cornea, mucous membranes of the urinary bladder and intestine, liver, kidney, and blood serum did not contain the antigen. Based on the results of disk electrophoresis in 5% polyacrylamide gels, 55%-81% alcohol extracts of the epidermal tissues, including the cornea and mucous membranes of the bladder, were of similar quantitative and qualitative composition. The composition differed from that of tissues of other origin. (15 refs.)

- 77-3340 **Effect of Immune Complexes on the Growth of Murine Tumors (Meeting Abstract).** (Eng.) Targowski, S. P. (Dept. Microbiology, Sch. Medicine, SUNY at Buffalo, Buffalo, NY 14214) Albini, B.; Abeyounis, C. J.; Milgrom, F. *Fed Proc* 36(3): 1204; 1977. (no refs.)



**77-3341 Transplantation Resistance of Syngeneic Mice Against a Drug-Treated Lymphoma Derived from Non-Immunogenic Tumor Line (Meeting Abstract).** (Eng.) Fioretti, M. C. (Inst. Pharmacology, Univ. Perugia, Italy 20014) Santoni, A.; Romani, L.; Taramelli, D.; Caprino, M. C.; Goldin, A.; Bonmassar, E. *Fed Proc* 36(3): 1260; 1977. (no refs.)

**77-3342 Experience with Heterotransplanted Human Tumors in the Nude Mouse (Meeting Abstract).** (Eng.) Sharkey, F. E. (Memorial Sloan-Kettering Cancer Center, New York, NY) Fogh, J. M.; Hajdu, S. I.; Fitzgerald, P. J.; Fogh, J. *Am J Pathol* 86(2): 29a; 1977. (no refs.)

**77-3343 Growth of Human Tumors in the Nude Mouse.** (Eng.) Poulsen, C. O.; Rygaard, J. In: *In Vitro Methods in Cell-Mediated and Tumor Immunity*. Bloom, B. R.; David, J. R., eds. (New York: Academic Press, Inc.): pp. 701-711; 1976.

Human tumor growth, using tumors from patients undergoing surgery in several hospitals, was assessed in nude mice. A total of 102 human malignant tumors of varied type, including adenocarcinoma of the gastrointestinal tract, melanoma, and epidermoid carcinoma, were transplanted in the mice. Male heterozygous carriers of the *nu* gene were crossed with noninbred NMRI/BOM-f females. The gene was transferred to three inbred strains of mice: BALB/c/A/BOM-f, C3H/bi/BOMf, and C57/BI/6/BOMf. The percentage of successful tumor graftings in the first transplant generation was 44, and 26/42 tumors were grown serially. After a latency period of 8-40 days, a nodule appeared at the inoculation site. Tumors grew as spherical nodules, increasing gradually in size until the death of the animal. The tumors grew locally, and the skin overlying the tumor was not adherent, as the tumor moved freely over the abdominal muscle layers. Mouse-grown tumors demonstrated a close similarity microscopically to the human donor material, even after 6 yr of transplantation. Two tumors (a Burkitt's lymphoma and a malignant melanoma) tended to infiltrate the sc fatty tissue and the muscle. The responsiveness of the tumors to anticancer agents was preserved after transplantation in nude mice. In conclusion, nude mice accept a high percentage of human solid tumors, the tumors demonstrate a constant and predictable growth pattern, and the human characteristics are preserved. (52 refs.)

**77-3344 Enhancement of Human Tumor Transplants in Nu/nu Mice (Meeting Abstract).** (Eng.) Helson,

L. (Sloan-Kettering Inst. for Cancer Res., New York, NY 10021) Helson, C.; Rubenstein, R.; Murphy, M. L. *Fed Proc* 36(3): 1256; 1977. (no refs.)

**77-3345 Hodgkin's Disease: A War Between T-Lymphocytes and Transformed Macrophages?** (Eng.) Kay, M. M. ((No affiliation given)) *Recent Results Cancer Res* 56: 111-122; 1976.

The interaction between autogenic T-cells and Reed-Sternberg (RS) cells was studied. Approx 200 RS were examined at intervals of 5, 10, 20, and 30 min. After 5 min of culture, it was observed that the tips of the T-cell microvilli were touching the lamellae (ruffles) and filopodia of the RS cells. About 6 T-cells were affixed to each RS cell. The membranes of the RS cells did not appear to have holes or gaps. After 10 min of culture, the T-cell microvilli were firmly affixed to RS cells; it appeared that the membranes of the RS cells were being pulled apart, creating interglobular gaps whose widths were 15 nanometers (nm) and holes with a width of 50-100 nm. After 20 min, the interglobular gaps were between 40-60 nm in width, and the membranes of RS cells were disintegrating. After 30 min, only loosely attached globular subunits around nuclei and scattered cytoplasmic organelles remained of the membranes of RS cells. The results suggest that the gaps and holes allow leakage of ions and influx of fluid, which result in target-cell lysis by osmotic swelling. In order to cytolyse RS cells, the number of T to target cells must be greater than three, or the T-cells can be attacked and destroyed. Occasionally, 'normal' macrophages and/or monocytes were attacked by lymphocytes. This might mean that the surface antigens are altered in Hodgkin's disease. It is suggested that macrophages of patients with Hodgkin's disease may have been infected with a virus that they are not able to digest after engulfment. (31 refs.)

**77-3346 Increased Reactivity to Lymphokines of Macrophages from Tumor-Bearing Mice (Meeting Abstract).** (Eng.) Ruco, L. P. (NCI, Bethesda, MD 20014) Meltzer, M. S.; Evans, C. H. *Proc Am Assoc Cancer Res* 18: 85; 1977. (no refs.)

**77-3347 Flow Cytometric Characterization of the Macrophage Response During Methylcholanthrene (MCA) Induced Tumor (Meeting Abstract).** (Eng.) Thornthwaite, J. T. (Univ. Miami Sch. Medicine, Miami, FL 33152) Sugarbaker, E. V. *Proc Am Assoc Cancer Res* 18: 200; 1977. (no refs.)

77-3348 **Direct Demonstration of Murine Thymus-dependent Cell Surface Endogenous Immunoglobulin.** (Eng.) Szenberg, A. (Walter and Eliza Hall Inst. Medical Res., Post Office Royal Melbourne Hosp., Parkville, Victoria 3050, Australia) Marchalonis, J. J.; Warner, N. L. *Proc Natl Acad Sci USA* 74(5): 2113-2117; 1977.

Anti-immunoglobulin reagents that visualize T-cell immunoglobulin (Ig) by immunofluorescence were developed using chickens as the source of antibody. Chickens were immunized by injection of mouse (Fab)<sub>2</sub> (2 mg/bird) and then bled 3 wk later. Immune sera were adsorbed on a column of mouse IgG-Sepharose 4B, and specific antibodies were eluted. The purified antibody consisted predominantly of 7S IgG(IgY), as assessed by polyacrylamide gel electrophoresis. These avian antibodies gave strong indirect immunofluorescence with murine thymus cells, peripheral T cells and T lymphoma cells. Normal spleen cells and spleen cells from CBA-nu/nu mice were also positive, indicating that B cells also react with this antibody preparation. In capping experiments, the Ig cap was shed and then reappeared, indicating that it is of endogenous origin. Nonlymphoid tumor cells of various myeloid types did not bind this reagent, even though they had avid Fc receptors. The capacity of chicken antibodies to bind to both B- and T-cell lymphomas was abolished by adsorption with myeloma-derived K chains coupled to Sepharose. The K antigenic determinant recognized by the chicken antibodies

may thus be different from that seen by mammalian antibodies. It is concluded that T lymphocytes and T lymphoma cells express and synthesize a surface Ig containing determinants that cross-react with B-cell-derived K chains. (24 refs.)

\* (Rev): 77-3031, 77-3032, 77-3033, 77-3034, 77-3035, 77-3036, 77-3062.

\* (Chem): 77-3095, 77-3136, 77-3172.

\* (Phys): 77-3217.

\* (Viral): 77-3232, 77-3240, 77-3241, 77-3244, 77-3256, 77-3263, 77-3264, 77-3265, 77-3267, 77-3279, 77-3284, 77-3295, 77-3298, 77-3299, 77-3300, 77-3302, 77-3304, 77-3305, 77-3320.

\* (Path): 77-3352, 77-3421, 77-3435, 77-3436, 77-3437, 77-3438, 77-3444, 77-3447, 77-3448, 77-3454, 77-3461, 77-3462, 77-3464, 77-3467, 77-3470, 77-3482, 77-3483, 77-3484, 77-3485, 77-3486, 77-3489, 77-3491.

\* (Epid): 77-3521, 77-3522.



## PATHOGENESIS

- 77-3349 **Rate-limiting Step in the Progression of Mouse Breast Tumors.** (Eng.) Prehn, R. T. (Jackson Lab., Bar Harbor, ME 04609) *Int J Cancer* 19(5): 670-672; 1977.

The incidence of breast tumors in C3H mice, Balb/c mice, and humans and the incidence of cervical cancer in humans were plotted as a function of age. In both strains of mice the incidence increases as a power of age until the eighth to ninth months. Thereafter, it remains virtually constant in C3H mice: about 46% of the animals at risk in each successive month develop cancer. Very late, in the 15th or 16th month, there is an apparent fall in incidence. This fall occurs earlier in Balb/c mice and tends to obscure the plateau of constant incidence. It is inferred that all hyperplastic nodules that will ever be at risk of becoming malignant complete their progression to a Stage I step immediately prior to malignancy by the eighth or ninth month of life. At this point the mature nodules await a further random event that transforms them to the neoplastic state. In contrast, the incidence of the two human cancers does not plateau at a certain age, but its rate of increase with age decreases dramatically at about age 40. (11 refs.)

- 77-3350 **Ultrastructural Studies on the Morphogenesis of Psammoma Bodies in Ovarian Serous Neoplasia.** (Eng.) Ferenczy, A. (Dept. Pathology, Jewish General Hosp., 3755 Cote St. Catherine Road, Montreal, Quebec, Canada H3T 1E2) Talens, M.; Zoghby, M.; Hussain, S. S. *Cancer* 39(6): 2451-2459; 1977.

Transmission electron microscope studies were carried out on psammoma bodies in two benign and seven malignant papillary serous neoplasms of the ovary. Ultrastructurally, psammoma bodies are composed of microcrystals similar to the calcium phosphate apatite crystals of bone. Formation of the bodies is initiated intracellularly, in both neoplastic epithelial cells and stromal histiocytes. The initial seeding site of apatite crystals is served by lipid-rich intracellular vesicles. These structures are produced in association with autophagocytosis in neoplastic epithelial cells and with heterophagocytosis of extracellular lipidic material in stromal histiocytes. Extracellular lipids presumably derive from dehiscent tumor tissue. The close relationship between larger intraepithelial calcific bodies and microfilaments suggests that the latter provide a supportive matrix for further intracellular calcification. Large extracellular psammoma bodies result from fused calcific bodies extruded from calcified cells. Mineralization of extracellular collagen fibers is not observed. These observations support the concept that psammoma bo-

dies in ovarian serous neoplasms are a consequence of dystrophic calcification associated with ischemia. (18 refs.)

- 77-3351 **Scanning Electron Microscopic Study of Prostatic Cancer.** (Eng.) Gaeta, J. F. (Natl. Prostatic Cancer Project, 666 Elm St., Buffalo, NY 14263) Berger, J. E.; Gamarra, M. C. *Cancer Treat Rep* 61(2): 227-253; 1977.

Changes occurring in 5 cases of benign prostatic hyperplasia and in 15 cases of prostatic adenocarcinoma of different degrees of differentiation were examined by scanning electron microscopy, which provides three-dimensional images of the tissues being studied. Prostatic hyperplasia was largely characterized by epithelial changes in the form of a significant increase in size and exaggeration of the pattern of normal acinar and ductal structures. A wide spectrum of surface changes that correlated well with histologic changes was observed in the carcinoma specimens. They were frequently composed of an epithelial population of cells that displayed a smooth surface with sharp angles and hexagonal shapes. Neoplastic cells consistently demonstrated a complete lack of villous structures, irregular shapes, and surface pits and crevices. The earliest alteration appeared to be the disruption of villous attachments that normally maintain cohesive organization of ductal and acinar epithelia. A second pathway of extension within the gland may consist of a gradual transformation of prostatic ducts as they are approached by the advancing edge of the tumor. (9 refs.)

- 77-3352 **Morphologic and Immunologic Studies of Human Prostatic Carcinoma.** (Eng.) Mickey, D. D. (Dept. Surgery, Urology Res., Box 3062, Duke Univ. Medical Center, Durham, NC 27710) Stone, K. R.; Stone, M. P.; Paulson, D. F. *Cancer Treat Rep* 61(2): 133-138; 1977.

Benign and malignant prostatic surgical specimens and tissue culture cells were analyzed ultrastructurally and immunohistochemically for the presence of viruses or viruslike particles. When 14 malignant and 18 benign prostatic hypertrophy (BPH) tissues were examined by transmission electron microscopy, viruses could not be identified with certainty in thin sections. Scanning electron microscopic examination of 24 BPH and 21 adenocarcinoma specimens revealed heterogeneous morphology. Normal, BPH, and neoplastic acinar cell surfaces could be classified as microvillous, ruffled, or bare. Incubation of tissues with crude collagenase removed the sup-

portive stroma without disrupting the gland structure itself. In a competition radioimmunoassays of 146 human urothelial tissues (71 tumorous, 75 nontumorous), 28% showed evidence of competition for p30 antigen, a component of C-type RNA viruses. Thirty-eight percent of the 18 adenocarcinoma specimens and 48% of the 42 BPH specimens were positive for the p30 core protein. The viral proteins may not have an oncogenic role, however, because activity can be demonstrated in both benign and malignant tissues. (18 refs.)

**77-3353 Familial Testicular Cancer in a Father (Bilateral Seminoma-Embryonal Cell Carcinoma) and Son (Teratocarcinoma): A Case Report and Review of the Literature.** (Eng.) Lapes, M. (Dept. Hematology-Oncology, Crozer-Chester Medical Center, 15th St. and Upland Ave., Chester, PA 19013) Iozzi, L.; Ziegenfus, W. D.; Antoniades, H.; Vivacqua, R. *Cancer* 39(5): 2317-2320; 1977.

The case reports of a father and son with testicular neoplasias, the fifth reported example of father/son familial testicular cancer, are presented. The histology of the father's tumor was seminoma with embryonal elements; he presented at age 34. His son, who presented at age 22, had teratocarcinoma in both the primary testicular tumor and metastatic retroperitoneal lymph nodes. This difference in tumor types was also apparent in 3/4 other reported father-son cases. The finding may reflect the multipotential nature of the germ cell giving rise to testicular neoplasia. No other factors predisposing to testicular cancer could be identified, including cryptorchidism or immunological deficiency. Patients and their families should receive appropriate genetic counseling regarding the potential hereditary nature of testicular cancer. (10 refs.)

**77-3354 On the Histology of Human Seminoma: Development of the Solid Tumor from Intratubular Cells.** (Eng.) Schultz, C. (Anatomisches Institut, Martinistrasse 52, 2000 Hamburg 20, W. Germany) Holstein, A. F. *Cancer* 39(3): 1090-1100; 1977.

Intratubular seminomas of the testis associated with tumors were studied by light and electron microscopy to obtain information about the earliest stage of emigration from the tubules and invasion of the interstitial space. Careful examination of numerous sections revealed that at this stage, neoplastic cells protrude into evaginations of the tubule covered by a basement membrane or directly invade that membrane. At the site of tumor cell emigration, the basement membrane is thickened and multilayered. Tumor cells devoid of basement membrane were found in the interstitium. Their transmigration through the basement membrane was never observed. Subsequent to the migration of seminoma cells, the tubules are smaller in diameter and contain only Sertoli cells. From

the observations, it is inferred that tumor cells in the interstitial tissue increase in number, form strands and lobules, and finally build up the solid tumor. (30 refs.)

**77-3355 Seminoma in Klinefelter's Syndrome with 47, XXY, 15s+ Karyotype.** (Eng.) Isurugi, K. (Dept. Urology, Univ. Tokyo Branch Hosp., Mejirodai, Bunkyo-ku, Tokyo 112) Imao, S.; Hirose, K.; Aoki, H. *Cancer* 39(5): 2041-2047; 1977.

An association of seminoma of the testis and Klinefelter's syndrome in a 32-yr-old man is reported. The patient was found to have seminoma of the right testis, which had been subjected to orchiopexy for cryptorchidism 14 yr earlier; the left testis was small and firm. Chromosomal analysis revealed a karyotype of 47, XXY, 15s+, with enlarged and fluorescent satellites on chromosome 15. The satellites were also found in the mother, two sisters, and one brother. Another sibling did not have satellites. Endocrine studies, the histology of the biopsied left testis, and dermatoglyphic analysis were compatible with Klinefelter's syndrome. This is believed to be the first reported case of seminoma associated with the syndrome. (22 refs.)

**77-3356 Carcinoma of the Gastric Stump: Clinical Experiences and Observations Over 15 Years.** (Ger.) Kienzle, H. F. (Chirurgische Klinik der Stadtischen Krankenanstalten, Moltkestrasse 14, D-7500 Karlsruhe 1, W. Germany) *Med Klin* 72(10): 399-405; 1977.

From 1960 to 1975, 29 patients were treated for carcinoma of the gastric stump following resection for benign disease. Of all gastric carcinomas, 2.38% were carcinomas of the stump. The patients were an av of 38.9 yr old at the time of resection for benign lesions and 60.6 yr old at the time of diagnosis of the carcinoma, for an av latency of 22.2 yr. The younger a patient is at the time of primary resection, the longer the latency period will be. The symptoms lasted 3.4 mo before presentation: the main complaints were epigastric pains, loss of wt, vomiting, and aversion for meat. The carcinomas were located at the anastomosis in 60%, at the cardia in 36%. Other locations were distributed over the entire gastric stump. Fourteen patients underwent gastrectomy with interposition of the jejunum, but the remainder were primarily inoperable or they were treated with prostheses. All types of carcinomas were found, predominantly adenocarcinoma (56%). Thirty-two percent of the patients died in the clinic; 17 patients survived an av of 8.5 mo after leaving the clinic. It is concluded that resection for ulcer is not a predisposing factor in the development of carcinoma of the gastric stump. A causal connection may be established when wide-range statistics, compiled under controlled and comparable conditions, are available. (42 refs.)



- 77-3357 **Ultrastructure of Intestinal and Diffuse Type Gastric Carcinoma.** (Eng.) Nevalainen, T. J. (Lab. Electron Microscopy, Univ. Turku, Kiinamyllynakku 10, SF-20520 Turku 52, Finland) Jarvi, O. H. *J Pathol* 122(3): 129-136; 1977.

The ultrastructural features of intestinal and diffuse type gastric carcinomas were described and compared to those of normal gastric mucosal cells and cells occurring in intestinal metaplasia of the gastric mucosa. Specimens from 56 resected stomachs comprised 28 intestinal adenocarcinomas, 14 diffuse carcinomas, 5 poorly differentiated solid carcinomas, and 9 nonneoplastic lesions. Electron microscopy provided further evidence of the structural similarity of gastric adenocarcinoma and intestinal metaplasia cells. The diffuse carcinoma cells closely resembled the goblet cells of intestinal metaplasia. The low superficial epithelium in the diffuse carcinoma contained a brush border of microvilli structurally identical to those in the brush border of the intestinal metaplasias and intestinal gastric carcinomas. Intracellular cysts with microvilli on the cyst wall were occasionally found in both types of carcinoma. Apparently, diffuse carcinoma is also a carcinoma derived from epithelium that has undergone metaplasia to the intestinal type. The demonstration of secretion in the intervening columnar cells in intestinal metaplasia deserves attention, as these cells, together with goblet cells, could be a source of tumor cells in diffuse carcinomas. (36 refs.)

- 77-3358 **Ultrastructural Features of Differentiation of Stomach Cancer Cells.** (Rus.) Rottenberg, V. I. (Dept. Pathological Anatomy Human Tumors, Oncological Scientific Center, Acad. Medical Sciences USSR, Moscow, USSR) *Arkhl Patol* 39(3): 32-39; 1977.

The results of an electron microscopic analysis of six stomach tumor specimens obtained at surgery are presented. There were 4 adenocarcinomas, 1 poorly differentiated carcinoma, and 1 signet ring cell carcinoma. One adenocarcinoma, which was 5 cm in diameter, consisted of several types of cells: mucoid cells, nondifferentiated cells, endocrine cells, surrounding cells, chief cells, and enterocytes. The five remaining tumors consisted of more or less homogeneous cell types. (21 refs.)

- 77-3359 **Histogenesis of Stomach Cancer and the Role of Intestinal Metaplasia in This Process.** (Rus.) Korlatsan, V. V. (Dept. Pathomorphology, Modavian Scientific Res. Inst. Oncology, Kishinev, USSR) *Arkhl Patol* 39(3): 39-44; 1977.

Histological examination was made of the biopsy specimens from 854 chronic gastritis patients who had been subjected to fibrogastroscopy. Superficial carcinomas were detected in 15 patients; the superficial carcinoma developed from a

chronic ulcer in 9 and from chronic gastritis in 6 cases. All the superficial carcinoma cases could be subdivided into two groups: in Group 1, tumor growth was observed only in the superficial third of the mucous membrane thickness of the stomach; in Group 2, tumors occupied from one-third to one-half of the mucous membrane thickness. Hyperplastic changes in the epithelium around the tumor were of two types: glands close to the tumor were covered with proliferating atypical epithelium that could undergo malignant transformation; glands far from the tumor were covered with proliferating but not atypical epithelium that could give rise to intestinal metaplasia. (16 refs.)

- 77-3360 **Adenocarcinoma of the Small Intestine in Crohn Disease Involving the Small Bowel.** (Eng.) Burbige, E. J. (Veterans Admin. Hosp., 150 Muir Road, Martinez, CA 94553) Bedine, M. S.; Handelsman, J. C. *West J Med* 127(1): 43-45; 1977.

The case history of a 43-yr-old man with a long history of Crohn's disease who subsequently developed adenocarcinoma of the small intestine is presented, and the literature is reviewed. The incidence of malignancy in patients with Crohn's disease has not been clearly established, but an association is indicated. (26 refs.)

- 77-3361 **Colon Carcinoma and the Cancer Family Syndrome.** (Eng.) Arndt, R. D. (Dept. Radiology, St. John's Hosp. and Health Center, 1328 22nd St., Santa Monica, CA 90404) Kositchek, R. J.; Boasberg, P. D. *JAMA* 237(26): 2847-2848; 1977.

The case histories of a 54-yr-old woman and her two children, a woman aged 37 and a man aged 32, are presented. The mother and the daughter each had adenocarcinoma of the colon while the son had an adenomatous polyp. All lesions were located essentially in the same region of the large bowel, indicating the involvement of hereditary factors. Closer screening of families of colon cancer patients is advised. (5 refs.)

- 77-3362 **Familial Polyposis Coli, a Report of One Family Pedigree.** (Jpn.) Matsumoto, T. (Matsumoto Gastrointestinal Hosp., 1 Tabame Hidaka-Cho Iruma-Gun, Saitama, Japan) Matsumoto, A.; Sekine, M.; Jinnai, T.; Yasui, E. *Stomach Intest* 12(4): 477-482; 1977.

Rectal or colonic cancer occurred in five members of the same family over three generations. Familial polyposis coli (FPC) was confirmed in three cases and strongly suspected

in the other two. The case histories of two of these members are presented: a 64-yr-old woman who was the daughter of the man who originally developed the disease and her 23-yr-old daughter. The main symptoms were intractable diarrhea often associated with hematorrhea. The 64-yr-old patient had rectal cancer and an abundance of small polyps in the stomach. An osteomalike lesion was found in the mandible, and a fist-sized soft tissue tumor was located in the left femoral region. The 23-yr-old woman had numerous small, hemispherical, adenomatous polyps in her large bowel. A villous tumor was observed in the transverse colon and a focal carcinoma with two cancerous lesions was found in the sigmoid colon. All cancerous lesions were histologically well-differentiated adenocarcinoma. It was concluded that FPC is a generalized disorder involving the large bowel, upper gastrointestinal tract, and other organs. (18 refs.)

- 77-3363 **Development of Squamous Cell Carcinoma in Chronic Anal Fistula.** (Ger.) Ritter, L. (Institut für Ärztliche Fortbildung, Klinik für Chirurgie II, Postfach 112, H-1389 Budapest, Hungary) Magyar, E. *Fortschr Med* 95(20): 1365-1367; 1977.

A 62-yr-old patient developed a squamous epithelial cell carcinoma at the site of an anal fistula that had followed an abscess. The fistula had been extirpated except for one branch that was electrocoagulated. The patient was symptom-free for 11 mo but then developed pain, bleeding, and fecal incontinence. The 3- x 3-cm growth was identified histologically. The tumor was electrocoagulated after the patient refused radical abdominal surgery or colostomy. He was irradiated once (2 fields, 3,000 R each) and then sent back to his home in South America with recommendations for surgical control and further radiotherapy. (25 refs.)

- 77-3364 **Evaluation of Cancer Risk in Chronic Ulcerative Colitis: Progress Report of a Prospective Study (Meeting Abstract).** (Eng.) Levin, B. (Univ. Chicago Hosp., Chicago, IL) Riddell, R. H. *Gastroenterology* 72(5/Part 2): 1088; 1977. (no refs.)

- 77-3365 **Atypical Sweat Duct Hyperplasia Accompanying Keratoacanthoma.** (Eng.) Santa Cruz, D. J. (Ohio State Univ. Coll. Medicine, Columbus, OH 43210) Clausen, K. *Dermatologica* 154(3): 156-160; 1977.

Atypical sweat duct hyperplasia is discussed based on 20 keratoacanthomas, 2 basal cell carcinomas, 2 granular cell tumors and 1 leiomyosarcoma. The etiology of the atypia is considered to be compression of the duct either in its dermal or intraepidermal portion. (13 refs.)

- 77-3366 **Invololution of Mucocutaneous Pigmentation of the Peutz-Jeghers Syndrome.** (Eng.) Keeling, P. W. (St. Thomas's Hosp., London SE1 7EH, England) Aston, N.; Anderson, H. J. *Br Med J* 1(6066): 949; 1977.

A 43-yr-old woman with Peutz-Jeghers syndrome experienced a disappearance of her mucosal and digital pigmentation and regression of an unresected small intestinal hamartoma following resection of three ileal polyps. Her daughter was also found to have a single bowel polyp, which was removed by colonoscopy. (4 refs.)

- 77-3367 **DNA Repair Characteristics and Skin Cancers of Xeroderma Pigmentosum Patients in Japan.** (Eng.) Takebe, H. (Radiation Biology Center, Kyoto Univ., Yoshida-Konocho, Sakyo-Ku, Kyoto 606, Japan) Miki, Y.; Kozuka, T.; Furuyama, J.; Tanaka, K.; Sasaki, M. S.; Fujiwara, Y.; Akiba, H. *Cancer Res* 37(2): 490-495; 1977.

Fifty Japanese xeroderma pigmentosum patients were examined for clinical and DNA repair characteristics. Skin cancers developed in 22 patients. Most of the patients without skin cancers were children, except for five older patients who had intermediate or nearly normal levels of DNA repair. All patients < 10 yr old had no or very low unscheduled DNA synthesis activity after UV light irradiation. Three genetic complementation groups, A, D, and E, and three variants were found. There were 21 Group A patients and no Group C patients; in Europe and the US, Group C patients are the most frequent. The large number of patients with low DNA repair capacities may account for the apparent high incidence of xeroderma pigmentosum in Japan. The age distribution of the cancer-bearing patients and their DNA repair characteristics suggest that almost all xeroderma pigmentosum patients will develop skin cancers unless their cells have nearly normal levels of DNA repair. (27 refs.)

- 77-3368 **Genetic Complementation Tests of Japanese Xeroderma Pigmentosum Patients, and Their Skin Cancers and DNA Repair Characteristics.** (Eng.) Takebe, H. In: *Fundamentals in Cancer Prevention. Proceedings of the 6th International Symposium, the Princess Takamatsu Cancer Research Fund, Tokyo, 1975.* Magee, P. N.; Takayama, S.; Sugimura, T.; Matsushima, T., eds. (Princess Takamatsu Cancer Research Fund, Tokyo, Japan): pp. 383-395; 1976.

In a group of 42 Japanese patients with xeroderma pigmentosum (XP), 23 were children under age 13. Among 22 who developed skin cancer (mostly on the face), the youngest were 5 yr old. Genetic complementation tests performed on the cells from 21 patients showed that 19 belonged to Group A, one to D and 1 to E. This distribution is markedly different from that in Europe and the United States where relatively



fewer are in Group A and many are in Group C. Unscheduled DNA synthesis (UDS) after UV irradiation was studied in cells derived from all of these XP patients. In the low repair group (less than 5% of normal), consanguinity was known in the families of 8 of the 25 patients. Among the 37 sibships in the total group, 11 represented first cousin marriages. Cells with 70-100% repair replication also showed reduced host-cell reactivation of UV-irradiated herpes simplex virus. Prevention of skin cancer in the "low repair" group, who usually develop cancer in the teens, might be very difficult. However, decisive diagnosis by UDS tests during infancy followed by protection from sun might prevent cancer development. The potential for prevention of cancer is greater in those with UDS of 50% or more, a group which tends to develop XP later in life. (32 refs.)

- 77-3369 **Membranous Basal Cell Adenoma of Parotid Gland, Dermal Cyndromas, and Trichoepitheliomas. Comparative Histochemistry and Ultrastructure.** (Eng.) Headington, J. T. (Dept. Pathology, 1335 E. Catherine St., Ann Arbor, MI 48109) Batsakis, J. G.; Beals, T. F.; Campbell, T. E.; Simmons, J. L.; Stone, W. D. *Cancer* 39(6): 2460-2469; 1977.

A study of a 70-yr-old man with basal cell adenoma of the parotid gland, dermal cylindromas and trichoepitheliomas suggests that these disorders may be the result of a single pleiotropic gene acting on ontogenetically similar stem cells. Furthermore, the basal cell adenoma may represent transformation of cells of the intercalated duct. (14 refs.)

- 77-3370 **Plastic Surgery for Basal Cell Carcinoma. A Clinical and Histopathological Study of Basal Cell Carcinomas.** (Eng.) Paavolainen, P. (Surgical Hosp., Kasarminkatu 11-13, SF-00130 Helsinki 13, Finland) *Ann Chir Gynaecol Fenn* 65(5): 345-351; 1976.

The clinical and morphological features in recurrent and non-recurrent basal cell carcinoma were compared in 70 patients with 79 carcinomas seen between 1968 and 1973. The group was composed of 38 men and 32 women; two patients had three tumors and five patients had two. The followup time varied from 1 to 34 yr. The primary tumor was ulcerative in 20 nonrecurrent and 20 recurrent cases and infected in 20 and 13 cases, respectively. More than half the tumors were >2 cm in diameter. Biopsy was the initial treatment in 30 tumors; 22 tumors received radiotherapy and only 7 were treated with wide excisions. Twenty tumors were referred primarily for plastic surgery. Tumors that were completely excised recurred in only 9.7%; 90.3% recurred when there was incomplete excision. In primary tumors without recurrences, there was a slightly higher amount of lymphocyte and plasma cell infiltration. The solid type tumor recurred most often (54.8%) followed by sclerosing (16.1%) and superficial (3.2%). (1 ref.)

- 77-3371 **Scar Carcinoma: Prognosis and Treatment.** (Eng.) Stromberg, B. V. (Dept. Surgery, Johns Hopkins Hosp., Baltimore, MD 21205) Keiter, J. E.; Wray, R. C.; Weeks, P. M. *South Med J* 70(7): 821-822; 1977.

A 30-yr experience with 31 cases of squamous cell carcinoma arising in scars is reviewed. The mean age of the patients was 58 yr. The av time from injury to diagnosis was 23 yr. The male to female ratio was 4:1. Tumors were well-differentiated in 23 cases and poorly differentiated in 8. The 3-yr survival rate was 94% for patients with well-differentiated lesions, but only 38% for those with poorly differentiated lesions that were more likely to metastasize. The implications of these findings are discussed in relation to therapy particularly regional lymph node dissection. (16 refs.)

- 77-3372 **Carcinoma on Old Frostbites.** (Eng.) Katsas, A. (Third Surgical Service, Evangelismos Hosp., Athens 140, Greece) Agnantis, J.; Smyrnis, S.; Kakavoulis, T. *Am J Surg* 133(3): 377-378; 1977.

Reports are presented of two men, aged 52 and 56 yr, who developed epidermoid carcinoma on frostbite scars of the feet. A review of the literature revealed only eight such cases. (4 refs.)

- 77-3373 **Primary Mucinous Carcinoma of Skin: Histochemistry and Electron Microscopy.** (Eng.) Headington, J. T. (Dept. Pathology, Univ. Michigan Medical Center, Ann Arbor, MI 48109) *Cancer* 39(3): 1055-1063; 1977.

Two primary mucinous carcinomas of skin were studied by histochemistry and by light and electron microscopy. Enzyme histochemistry showed a pattern of reactivity similar to that found in eccrine secretory epithelium, eg, abundant phosphorylase and limited nonspecific esterase and acid phosphatase activities. Mucin histochemistry substantiated previous reports of probable sialomucin formation. Electron microscopy revealed a highly differentiated neoplasm with a mode of mucin secretion similar to that observed in the dark (mucinous) cell of the eccrine coil. The natural history of mucinous carcinoma of skin indicates that although local growth is the rule, lymph node metastasis may occur. (19 refs.)

- 77-3374 **On Histogenesis and Mechanism of Regression of Congenital Angiomas of the Skin in Children (Electron Microscopy Study).** (Rus.) Lebkova, N. P. (Central Scientific Res. Lab., Central Postgraduate Medical Institute, Moscow, USSR) Kodrian, A. A. *Arkh Patol* 39(3): 44-51; 1977.

An ultrastructural study of hemangiomas of the skin indicated that the tumor cells were similar to their ancestor endothelial cells. The ultrastructure of these cells is presented; many underwent fibrosis resulting in destruction of the tumor cells. (23 refs.)

**77-3375 Intra-endothelial Tubular Aggregates in Malignant Atrophic Papulosis (Degos' Disease).** (Eng.) Bleehan, S. S. (Rupert Hallam Dept. Dermatology, Hallamshire Hosp., Sheffield S10 2JF, England) *Clin Exp Dermatol* 2(1): 73-74; 1977.

It is suggested that the binding of intraendothelial tubular aggregates in patients with malignant atrophic papulosis could be the result of an alteration of the endoplasmic reticulum by a virus. These aggregates have also been found in other patients with skin diseases. (8 refs.)

**77-3376 Chronic Dermatitis and Cutaneous Squamous Cell Carcinoma in the Beagle Dog.** (Eng.) Hargis, A. M. (Collaborative Radiological Health Lab., Foothills Campus, Colorado State Univ., Fort Collins, CO 80523) Thomassen, R. W.; Phemister, R. D. *Vet Pathol* 14(3): 218-228; 1977.

The development of inflammatory and proliferative lesions in nonpigmented, sparsely haired abdominal, inguinal, preputial, and scrotal skin of beagles was studied. The dogs were housed at a moderately high altitude in a smog-free area with abundant sunshine. The area in which they lived was located outside on reflective gravel. Most of the dogs spent the daylight hours in full sunshine; they frequently rested on their backs with their ventral surfaces exposed to the sun. Of the 1,680 beagles studied, chronic dermatitis developed in 397 by the time they were 5.5 yr old. Twenty-six of 55 other beagles had developed these lesions by 12 yr of age. Squamous cell carcinomas developed in the dermatitis sites in 8/397 younger and in 5/26 older beagles. Circumstances suggest that solar radiation is involved in the pathologic process. In one dog the solar-induced carcinoma in the skin metastasized to the lung. (21 refs.)

**77-3377 The Value of Biological Investigations of Cultured Cells from Benign Pigmented Nevi.** A Preliminary Study. (Fre.) Aubert, C. (Unite 119 I.N.S.E.R.M., 27, bd Lei-Roure, 13009 Marseille, France) Amar, R.; Rouge, F.; Bureau, H. *Ann Chir Plast* 21(4): 271-276; 1976.

The biological, biochemical, and ultrastructural investigations of 17 pigmented nevus cell cultures obtained from seven patients are described. The medium contained 5-S-cysteinyldopa in three cultures, and this substance was also

detected in the cells in two of these cultures. Active cell division was observed in all cultures. The third subcultures of three cultures were treated with a conditioned medium from human malignant melanomas, containing a substance responsible for the malignant transformation of fibroblastoid cells. The treatment caused no changes in two cultures, but cells morphologically similar to those found in malignant melanoma cultures appeared in the third. However, this culture did not survive after the fourth cell division, which indicates the persistence of malignancy. Ovoid, fusiform, and fibroblast-type cells were found in the pigmented nevus cell cultures. An ultrastructural study of two cultures revealed reticular structures similar to those found in fibroblastoid cells from primary cultures of primary malignant melanoma in some cells. (6 refs.)

**77-3378 Extraskkeletal Ewing Sarcoma: An Ultrastructural Study.** (Eng.) Wigger, H. J. (Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, NY 10032) Salazar, G. H.; Blanc, W. A. *Arch Pathol Lab Med* 101(8): 446-449; 1977.

A nonosseous pelvic sarcoma in a 13-yr-old girl was studied by electron microscopy to demonstrate its relationship to Ewing sarcoma of bone. The girl was hospitalized because of severe periumbilical and right, lower-quadrant abdominal pain of 2 days' duration. This followed a 15-mo history of increasing urinary frequency and recent nocturia. Radiological studies showed displacement and obstructive compression of the bladder, but no evidence of bone involvement. Exploratory laparotomy revealed bloody ascites and a right-sided pelvic mass about 10 cm in diameter, which was resected. Upon electron microscopy, the diameter of the tumor cells was found to be about 12  $\mu$ m and that of the nuclei, 8-9  $\mu$ m. The cell outline was dependent on the surrounding cells. Many cells contained large empty areas and distended vacuoles delimited by a single membrane, which might belong to abnormal Golgi complexes or smooth endoplasmic reticulum. The ultrastructural characteristics were very similar to those of osseous examples (as shown in a table), except for the relative paucity of glycogen, the immaturity of the tumor cells, and the absence of mature or secondary cells. The features of this sarcoma were consistent with a very immature mesenchymal tumor, but they disclosed no evidence as to the site of origin of the tumor cell or to the direction of its potential differentiation. (9 refs.)

**77-3379 Elastofibroma, a Clinicopathological Study of 21 Cases.** (Jpn.) Nagamine, N. (Dept. Surgery, Sch. Health, Ryukyu Univ., Ryukyu, Japan) Miyagi, Y.; Endo, I.; Sho, Y.; Nohara, Y.; Itoh, E. *Jpn J Cancer Clin* 23(3): 203-213; 1977.

Twenty-one cases of elastofibroma, 20 in the back and 1 in the left olecranon, are presented. The patients ranged in age from 50 to 88 yr, and all had engaged in severe physical labor



for 15 to 61 yr. Of the 21 cases, all but 1 occurred in women. There were two instances of mother-daughter pairs, and two other patients had other suspected cases in their families. The lesions were located in the subscapular region in 20 cases: 13 bilateral, 5 right, and 2 left. Cystic components resembling synovial cysts were found in the tumor in three cases of elastofibroma of the back. (12 refs.)

- 77-3380 Metaplastic Squamous Cell Carcinoma of Bronchus Simulating Giant Cell Tumor of Bone.** (Eng.) Oyasu, R. (303 E. Superior St., Chicago, IL 60611) Battifora, H. A.; Buckingham, W. B.; Hidvegi, D. *Cancer* 39(3): 1119-1128; 1977.

A 57-yr-old man underwent a right pneumonectomy for a bronchogenic carcinoma following bronchoscopy and bronchial biopsy. The tumor was a polypoid mass arising from the lower lobe bronchus. It was characterized microscopically by mononuclear cells mixed with randomly distributed multinucleated giant cells similar to those seen in giant cell tumor of bone. Portions of the tumor showed typical squamous cell and spindle cell carcinoma. Based on light and electron microscopy findings, the case represents a metaplastic squamous carcinoma with mesenchymal cell differentiation. A hypothesis on the histogenesis of pleomorphic carcinomas is presented. Problems of the histological diagnosis of these tumors are emphasized. (37 refs.)

- 77-3381 Intranuclear Inclusions in a Giant Cell Tumor of Bone. Demonstration by Electron Microscopy.** (Fre.) Le Charpentier, Y. (Service Central d'Anatomie et de Cytologie Pathologiques, Faculte de Medecine Cochin-Port-Royal, 27, rue du Fg St-Jacques, F 75674 Paris Cedex 14, France) Le Charpentier, M.; Forest, M.; Daudet-Monsac, M.; Lavenue-Vacher, M. C.; Louvel, A.; Sedel, L.; Abelanet, R. *Nouv Presse Med* 6(4): 259-262; 1977.

A biopsy specimen from a giant cell tumor of the femur of a 30-yr-old man was studied ultrastructurally. A high proportion of the cells contained numerous filamentous inclusions within the nucleus in addition to the elements normally present. There were no membranes around the filaments, which formed a group about the size of a nucleolus, and they were not in contact with the other nuclear structures. The filaments, which were uniform in size (120-150 Å), appeared to be tubules with a clear center of about 60 Å. Similar structures are observed in both the nuclei and cytoplasm of the osteoclasts in Paget's disease. (25 refs.)

- 77-3382 Primary Osteosarcoma of Bone. A Clinicopathologic Investigation of 243 Cases, with Necropsy Studies in 54.** (Eng.) Uribe-Botero, G. (Dept. Pathology, Univ. Texas System Cancer Center, M.D. Anderson

Hosp. and Tumor Inst., Houston, TX 77030) Russell, W. O.; Sutow, W. W.; Martin, R. G. *Am J Clin Pathol* 67(5): 427-435; 1977.

A total of 243 patients (133 men, 110 women) with pathologically verified primary osteosarcomas of bone seen between 1950 and 1974 were investigated retrospectively. The study was undertaken to provide baseline information about patients treated prior to the use of routine adjuvant chemotherapy in the management of osteosarcoma. Osteosarcoma had its highest incidence in the second decade of life (60.9%), followed by the third (9.5%) and first (8.2%) decades. The femur was the most frequent site of the primary lesion (121 cases), followed by the tibia (47 cases). Survivals were directly attributable to surgical procedures, including resection of pulmonary metastases. The 3-yr survival rate for the series was 21.7%, with only 12.6% surviving 5 yr. The histologic pattern reproduced that of the primary lesion in all metastases; lymph node metastases were noted in only four cases, but pulmonary metastases were seen in all but one case. Among the three most frequent sites of occurrence, in terms of 3-yr survival, lesions originating in the humerus had the highest survival rate (19%), followed by the tibial and femoral lesions (14%). Patients with osteoblastic tumors had the poorest survival rate, followed by those with chondroblastic lesions. Individuals with primary lesions in the facial bones had the highest survival rate. (11 refs.)

- 77-3383 Localization of Alkaline Phosphatase in Subcellular Structures of Human Osteogenic Sarcomas.** (Eng.) Sokolova, I. N. (Dept. Pathological Anatomy and Human Tumors, Oncological Res. Center, Acad. Medical Sciences USSR, Moscow 115478, USSR) Litvinova, L. V. *Folia Histochem Cytochem (Krakow)* 14(4): 239-241; 1976.

An electron microscope study of human osteogenic sarcomas revealed that alkaline phosphatase was localized in the plasma membrane and processes, the perinuclear space, the endoplasmic reticulum, and, occasionally, around the lipid granules. The activity of the enzyme, which accumulated in the form of large granules and their aggregations, was particularly high in the plasma membrane and its processes. (7 refs.)

- 77-3384 Skeletal Metastases: Relationship of Bone Destruction, Osteoclast Activation and Prostaglandins (Meeting Abstract).** (Eng.) Galasko, C. S. (Orthopaedic Unit, Royal Postgraduate Medical Sch., Hammersmith Hosp., London W12 0HS, England) Bennett, A. *Br J Cancer* 35(2): 253-254; 1977. (no refs.)

- 77-3385 Changes in Bone and Bone Marrow of Rabbits Bearing the VX-2 Carcinoma: A Comparison of Local and Distant Effects.** (Eng.) Hough, A. (Dept. Pathology, Sch. Medicine, Vanderbilt Univ., Nashville, TN 37232)

Seyberth, H.; Oates, J.; Hartmann, W. *Am J Pathol* 87(3): 537-552; 1977.

The VX-2 carcinoma of New Zealand white rabbits, which originated in a virus-induced papilloma, was studied morphologically in 32 animals with respect to the osseous effects induced by the injection (1g, im) and subsequent growth of the tumor in the soft tissues for 1-5 wk. Although more severe changes were noted in the bones of tumor-bearing than in those of non-tumor-bearing limbs, effects could be seen in both experimental situations. Severe bone marrow hyperplasia developed in 29/32 tumor-bearing animals. A marked increase in the number of osteoclasts over control rabbit bones was observed along the surface of bones near and distant from the tumor. Resorptive changes were also noted in the cortex of the tumor-bearing rabbits. The increase in osteoclasts was related chronologically to the development of hypercalcemia, and it was proportional to the degree of hypercalcemia at the time the animals died. The number of osteoclasts was inversely proportional to the serum creatinine level. The number of osteoclasts decreased in the later stages of the disease. These changes may be the morphologic expression of humoral hypercalcemia accompanying the VX-2 carcinoma. (39 refs.)

**77-3386 An Osteosarcoma Cell and Matrix Retained Morphogen for Normal Bone Formation.** (Eng.)

Urist, M. R. (UCLA Bone Res. Lab., Rehabilitation Center, 1000 Veteran Ave., Room A3-34, Los Angeles, CA 90024) Nakata, N.; Felser, J. M.; Nogami, H.; Hanamura, H.; Miki, T.; Finerman, G. A. *Clin Orthop* (124): 251-266; 1977.

Two established transplantable osteosarcomas, Dunn and Ridgway, were injected sc (1.0 mm<sup>3</sup>) into susceptible CBA and AKR mice, respectively. Weanling 3-wk-old CBA mice were significantly more susceptible to tumor growth than the older recipients (4-20 wk). Histological examination of both osteosarcomas indicated the development of incompletely calcified, disorganized bone with no inclusion of cartilage or bone marrow. The predominant Dunn cell type, a malformed disorganized osteoblast, secreted excess alkaline phosphatase that increased with time. Implants of freeze-dried Dunn osteosarcoma were resorbed and replaced by normal cartilage, bone, and bone marrow, but implants of freeze-dried Ridgway osteosarcoma were replaced by a fibrous scar. Disaggregated Dunn cells synthesized a collagen-resistant, trypsin-labile, cell surface bone morphogen that also was found in the tumor matrix stroma. This normal bone morphogenetic protein initiates morphogenesis; its possible use to modify tumorigenesis warrants investigation. (46 refs.)

**77-3387 Histopathology of Phacomatoses.** (Fre.) Gontier, M. F. (No affiliation given) Piussan, Ch.; Risbourg, B.; Petit, J.; Reguet, C.; Boudailiez, B. *Med Infant (Paris)* 84(3): 231-246; 1977.

Histogenic studies on the ectoblast, mesoblast, and entoblast are reviewed in connection with the genesis of phacomatosis. Phacomatosis may be due to hamartomatosis of the neural crest and the hamartoma cells may be present at birth. Hamartoma cells are arrested during their proliferative stage, and their blastomatous development begins later, in the well-differentiated normal tissue in which they are located. The blastomatous tendency cannot be explained by faulty migration of the cells alone. Clinical observations appear to agree with the hypothesis on the early perturbation of germ layer development, especially that of the neuroectodermal layer and the neural crest. The common embryonal origin of phacomatosis is confirmed by the fairly high frequency of intermediary forms among the different types of phacomatoses that are often observed in the same family. (10 refs.)

**77-3388 The Ultrastructure of Pulmonary Hamartoma.**

(Eng.) Stone, F. J. (Dept. Pathology, New York Medical Coll., Basic Sciences Building, Valhalla, NY 10595) Churg, A. M. *Cancer* 39(3): 1064-1070; 1977.

An ultrastructural investigation of pulmonary hamartomas, which appear to arise de novo in middle-aged or elderly patients, was conducted. The epithelial cells corresponded to those lining the distal bronchioles and alveoli of adult lungs. Certain stromal and epithelial features resembled the structure of fetal lung. (17 refs.)

**77-3389 Coexistence of Bronchogenic Carcinoma and Gaucher Disease (Letter to Editor).** (Eng.)

Tsung, S. H. (Milwaukee, WI) Cotes, E. *Arch Pathol Lab Med* 101(1): 56; 1977.

The case history of a 56-yr-old man with bronchogenic carcinoma and Gaucher disease is presented. Although there was not necessarily a causal relationship between these diseases, a study of the function of Gaucher cells and immune competence may indicate some etiologic role. (2 refs.)

**77-3390 Growth Analysis of a Laryngeal Fibrosarcoma.**

(Jpn.) Matsui, T. (Dept. Otolaryngology, Kyoto Prefecture Univ. Medicine, Kyoto, Japan) Yanohara, K.; Ohshima, W.; Yamamichi, I.; Saito, H.; Mizukoshi, O.; Ashihara, T.; Takeoka, O. *Jpn J Cancer Clin* 23(3): 214-219; 1977.

The clinical course of a 75-yr-old man with laryngeal fibrosarcoma was studied over a 14-mo period. Radiographic measurements of tumor volume were used to calculate the growth rates of the metastatic lung tumors. The growth rates of the metastatic tumors were nearly exponential during the observation period. The av doubling time was 13.8 days. The estimated date of occurrence of tumor metastasis correlated



with the date of surgical intervention. After the occurrence of metastasis, the incipient growth stage was longer and the incipient growth speed was faster than those observed in the advanced growth stage. (15 refs.)

- 77-3391 **Malignant Transformation of Laryngeal Papillomas.** (Jpn.) Ishii, H. (Dept. Otorhinolaryngology, Gunma Univ. Sch. Medicine, Gunma, Japan) Kawabata, I. *J Otolaryngol Jpn* 80(3): 40-46; 1977.

Thirty-two cases of laryngeal papilloma, 22 in males and 10 in females, were treated over a 14-yr period. The patients ranged in age from 10 to > 60 yr; 10 of the patients were in their sixties. The lesions appeared hyperkeratotic upon laryngoscopic examination. Patients with localized-type papilloma were completely cured by surgery. Diffuse-type papilloma was resistant to both radiotherapy and chemotherapy. Malignant transformation occurred in five patients subjected to long-term follow-up studies. The interval between the onset of symptoms and malignant transformation was from 4 to 9 yr. Laryngectomy was performed on all patients, and they all survived without recurrence or metastasis. (19 refs.)

- 77-3392 **Follow-up Studies in Oral Leukoplakia.** (Eng.) Banoczy, J. (Dept. Conservative Dentistry, Semmelweis Medical Univ., H-1088 Budapest, Mikszath Kalman ter 5, Hungary) *J Maxillofac Surg* 5(1): 69-75; 1977.

Clinical and histopathological findings gained during a follow-up study of 670 oral leukoplakias registered over a 30-yr period (1946-1976) are presented. This group includes 40 cases of oral cancer originating from leukoplakia. Dysplasia was noted in 120/500 leukoplakia cases examined histologically. Carcinoma subsequently developed in 13% of the dysplasia cases. Leukoplakia was prevalent in the age group 51-60 yr, carcinoma in the age group 61-70 yr. This age group difference suggests that there is a latency period between the beginning of leukoplakia and the diagnosis of carcinoma, and it emphasizes the importance of long-term follow-up examinations. A male-female ratio of 3.2:1 was noted in the leukoplakia group, and a 1.9:1 ratio was noted in the carcinoma group. The sites of predilection for malignant transformation and dysplasia were the tongue and the lips. *Candida albicans* infection and the simultaneous existence of several etiological factors seemed to play a significant role in malignant transformation. Erosive leukoplakia showed the highest risk, with cancer occurring in 25% of the cases. (25 refs.)

- 77-3393 **Plasma Vitamin 'A' and Carotene Levels in Squamous Cell Carcinoma of Oral Cavity and Oropharynx.** (Eng.) Ibrahim, K. (Res. Cell, Pakistan Medical Res. Council, Jinnah Postgraduate Medical Centre, Karachi, Pakistan) Jafarey, N. A.; Zuberi, S. J. *Clin Oncol* 3(2): 203-207; 1977.

Plasma vitamin A and B carotene levels were estimated in 124 men and 79 women (22-80 yr old) with squamous cell carcinoma of the oral cavity and oropharynx and in 112 controls matched for sex and age. Most of the women were in their fourth, fifth, and sixth decades, and most of the men were in their fifth and sixth decades. The av values for carotene and vitamin A were 39.5 and 21.5  $\mu\text{g}\%$  in the patients and 61.5 and 39.67  $\mu\text{g}\%$  in the controls, respectively. Low levels of B carotene were found in 55% of the patients and 17% of the controls, and deficient or low levels of plasma vitamin A were found in 51% and 3%, respectively. Vitamin A deficiency may have some etiological significance in the increased frequency of oropharyngeal malignancies in developing countries like Pakistan. (37 refs.)

- 77-3394 **Characterization of a Spontaneous Esophageal Squamous Cell Carcinoma from a Rhesus Monkey (*Macaca mulatta*) and the Establishment of an Epithelial Cell Line (Meeting Abstract).** (Eng.) Neubauer, R. H. (Frederick Cancer Res. Center, Frederick, MD 21701) Hopkins, R. H.; Valerio, M. G.; Gonda, M. A. *In Vitro* 13(3): 174; 1977. (no refs.)

- 77-3395 **The Nature of Spindle Cells in Pleomorphic Adenomata: Ultrastructural Observations on Two Nasal Tumors (Meeting Abstract).** (Eng.) Sidhu, G. S. (New York Univ. Sch. Medicine, New York, NY) Vuletin, J. C. *Lab Invest* 36(3): 358; 1977. (no refs.)

- 77-3396 **In Vivo Incorporation of  $^3\text{H}$ -Choline in Murine Pulmonary Adenoma Cells: An Electron Microscopic Radioautographic Study.** (Eng.) Frasca, J. M. (Lab. Senior Medical Investigator, Veterans Admin. Hosp., East Orange, NJ 07019) *Exp Mol Pathol* 26(2): 290-301; 1977.

The in vivo incorporation of  $^3\text{H}$ -choline in the tumor cells of urethane-induced pulmonary adenomas of male A/He mice was examined by electron microscopic radioautography. The smallest lesion in the urethane-treated mice was recognized as a proliferation of type II cells. Most morphological differences in type II cells were encountered in tumors that appeared papillary and were > 2.0 mm in diameter. (27 refs.)

- 77-3397 **A Case of Amylase Producing Lung Cancer. Ultrastructural and Biochemical Studies.** (Jpn.) Yokoyama, M. (Third Dept. Internal Medicine, Sapporo Medical Coll., Sapporo, Japan) Natsuizaka, T.; Nakahashi, M.; Anzai, T.; Kikutsi, K.; Moriyama, Y.; Narita, A.; Kasagi, A. *Jpn J Cancer Clin* 23(3): 232-239; 1977.

Electron microscopic and biochemical studies were made in a case of lung cancer with elevated amylase activity in the

serum, urine, and tumor tissue. Ultrastructural studies showed that zymogen granules in the tumor cytoplasm were associated with postnatal or immature amylase granules in the salivary glands. The molecular wt and electrophoretic patterns of the tumor tissue amylase resembled those of salivary gland amylase, but the tumor tissue amylase was composed of a glycoprotein containing sialic acid. The origin of amylase-producing lung cancer was discussed. (20 refs.)

77-3398 **The Ferruginous Body Contents of Lungs at Autopsy in Boston, 1928-1932.** (Eng.) Gordon, P. (Mallory Inst. Pathology, Boston City Hosp., Boston, MA) Rosen, P. P.; Savino, A. *Acta Cytol (Baltimore)* 20(6): 521-524; 1976.

Lung tissues from autopsies performed from 1928 through 1932 in Boston were quantitatively studied by chemical digestion and microfiltration to determine if there has been an increase in pulmonary ferruginous body (fibrous material, including asbestos, coated with an iron-protein complex) content. Most of the 28 patients whose tissues were studied had died of nonneoplastic diseases and only 1/5 with cancer had primary pulmonary carcinoma. Ferruginous bodies were found in 11/28. These patients were 17-76 yr old, and the male/female ratio was 5/6. A similar age distribution was found among the 17 patients with no ferruginous bodies, and there was also a predominance of females in that group. Histologic sections of lung tissue from cases with and without the typical ferruginous bodies were not significantly different. The absence of ferruginous bodies in 61% of the patients and the finding that none of the samples contained over 20 such bodies is in contrast to recent studies, in which ferruginous bodies were found in 90%-100% of the lungs examined. These results suggest that there may have been an increased exposure over the past several decades to respirable fibrous materials that can be converted to ferruginous bodies. (15 refs.)

77-3399 **The Significance of Tuberculosis and Chronic Pneumonias as Precursors of Pulmonary Carcinoma.** (Rus.) Braude, V. I. (Dept. Pathomorphology, Moscow Scientific Res. Inst. Tuberculosis, Russian SSR Ministry Public Health, Moscow, USSR) *Sov Med*(12): 111-115; 1976.

The results of post-mortem examinations performed during 1965-1973 on 4,245 patients are presented. Pronounced pulmonary alterations were observed in 770 patients (454 had tuberculosis, 307 had chronic nonspecific inflammatory pulmonary disease and 9 had posttuberculous changes and chronic pneumonia). Cancer of the lung was observed in 110 patients (83 men, 27 women, aged 23-88 yr); among them 39 had no prior history of lung lesions, 22 had tuberculosis and 49 had nonspecific inflammatory pulmonary disease. Thus, incidence of the bronchogenic cancer was significantly higher among the patients who had preceding pulmonary diseases (9.22%) than among those who had healthy lungs (1.12%).

Patients with tuberculosis were found to have lung cancer less frequently than the patients with nonspecific pulmonary diseases (20% and 44.5%, respectively). (18 refs.)

77-3400 **Cell Aggregates in Malignant Mesothelioma.** (Eng.) Whitaker, D. (Dept. Pathology, Univ. Western Australia, Sir Charles Gairdner Hosp., Perth Medical Centre, Shenton Park, Western Australia) *Acta Cytol (Baltimore)* 21(2): 236-239; 1977.

The cytologic and ultrastructural morphology of cell aggregates found in 12 cases of malignant mesothelioma and 80 cases of metastatic carcinoma is discussed. Cell aggregates were frequently noted in smears made directly from an effusion due to malignant mesothelioma. The aggregates varied in size and shape, but were often rounded. Cell-block analyses showed that in 4/12 cases the cell aggregates contained a small amount of central, amorphous, eosinophilic material identified as collagen. This is statistically significant ( $p > 0.01$ ) when compared with the incidence of collagen core in only 2/80 cases of effusion due to metastatic carcinoma. These findings may be of diagnostic value in the cytologic evaluation of effusions when malignant mesothelioma is suspected. (15 refs.)

77-3401 **A Scanning Electron Microscopic Study of Diffuse Mesothelioma and Some Lung Carcinomas (Meeting Abstract).** (Eng.) Dionne, G. P. (Dept. Pathology, Royal Victoria Hosp., Montreal, P. Q., Canada) Wang, N. S. *Lab Invest* 36(3): 356; 1977. (no refs.)

77-3402 **An Ultrastructural Study of Lung Tumors (Meeting Abstract).** (Eng.) Kandawalla, N. M. (Veterans Admin. Hosp., Tampa, FL 33612) Kasnic, G.; Azar, H. A. *Lab Invest* 36(3): 342; 1977. (no refs.)

77-3403 **Isolated Trigeminal Neuropathy. An Unusual Complication of Carcinoma of the Lung.** (Eng.) Delaney, P. (Dept. Neurology, Georgetown Univ. Hosp., 3800 Reservoir Road NW, Washington, DC 20007) Khoa, N.; Saini, N. *JAMA* 237(23): 2522-2523; 1977.

The case history of a 62-yr-old man is presented in which trigeminal neuropathy was due to metastasis of carcinoma of the lung to the trigeminal ganglion. Cranial nerve examination revealed abnormalities in all three divisions of the right trigeminal nerve. The patient died shortly after treatment. Autopsy showed oat cell carcinoma of the lung with metastases to the liver, spleen, adrenal glands, kidneys, left ureter, lymph nodes, and vertebrae. The right trigeminal ganglion was enlarged and replaced by firm, whitish tumor tissue and was infiltrated by metastatic carcinoma. (10 refs.)



- 77-3404 The Histogenesis and Development of Pulmonary Tumorlets.** (Eng.) Ranchod, M. (Dept. Pathology, Stanford Univ. Medical Center, Stanford, CA 94305) *Cancer* 39(3): 1135-1145; 1977.

A lung surgically removed from a patient with oat cell carcinoma contained multiple tumorlets and showed extensive Kultschitsky cell proliferation of the bronchial and bronchiolar mucosa. Light and electron microscopy showed that pulmonary tumorlets arise from focal areas of bronchial and bronchiolar Kultschitsky cell proliferation that may advance to luminal obliteration. The pulmonary parenchyma is involved by extension of these newly proliferated cells along the terminal branches of the bronchiolar tree or by penetration of the bronchial or bronchiolar wall; the latter process evokes a striking proliferation of connective tissue that forms the matrix in which the cells of some fully developed tumorlets are embedded. Because of striking morphologic similarities between tumorlets and spindle cell carcinoid tumors and the proved origin of tumorlets from pulmonary Kultschitsky cells, it is suggested that the more complete and histogenetically acceptable term "carcinoid tumorlet" be used to distinguish this lesion from other forms of epithelial proliferations in the lung. (46 refs.)

- 77-3405 Morphological Studies on the Development of Tracheal Epithelium in the Syrian Golden Hamster. III. Electron Microscopy: Early Ciliogenesis.** (Eng.) Emura, M. (Abteilung für Experimentelle Pathologie, Medizinische Hochschule, Karl-Wiechert-Allee 9, D-3000 Hannover-Kleefeld, W. Germany) Reznik-Schuller, H.; Mohr, U. *Z Versuchstierkd* 19(1-2): 47-51; 1977.

The morphology of tracheal epithelial cells of Syrian golden hamsters at early stages of development was examined electron microscopically. It has previously been suggested that the susceptibility of the epithelial cells to the carcinogen diethylnitrosamine might be correlated with the state of differentiation of the target cells. Several cells seen on the 13th prenatal day were characterized by a paucity of granular endoplasmic reticulum and by the presence of fibrogranular masses in the luminal cytoplasm. These cells were considered presumptive ciliated cells. (11 refs.)

- 77-3406 Pleural Mesothelioma.** (Pol.) Srednicka-Zajac, D. (Klinika Ftyzjopulmonologiczna Instytutu Chorob Wewnętrznych AM, ul. Jaczewskiego 8, 20-950 Lublin, Poland) Szarewicz-Adamczyk, W. *Wiad Lek* 30(3): 169-174; 1977.

Six cases of mesothelioma (3 men and 3 women aged 19-67 yr) that were observed from 1972 to 1974 are reported. Four of the tumors were diffuse and two were localized. Two were diagnosed during operation, three by cytological examination of the pleural exudate, and one by needle biopsy. Two patients had occupational contact with asbestos. All of the patients showed the following symptoms: pain in the affected

part of the chest, dyspnea, dry cough, and progressing cachexia. The only effect of chemo- and radiotherapy was a slight prolongation of survival. An increase in the incidence of lung tumors within 10-20 yr is predicted because of the prevailing contact with asbestos and its derivatives. (15 refs.)

- 77-3407 The Fine Structure of Large Cell Undifferentiated Carcinoma of the Lung: Evidence for its Relation to Common Adeno- and Squamous Cell Carcinomas (Meeting Abstract).** (Eng.) Churg, A. (Dept. Pathology, Univ. Chicago Hosp. and Clinics, Chicago, IL 60637) Warnock, M. L. *Lab Invest* 36(3): 334; 1977. (no refs.)

- 77-3408 Multiple Congenital Myofibroblastic Hamartomas (Subdermal Fibrous Hamartomas) of Infancy: Ultrastructural and Immunofluorescent Studies (Meeting Abstract).** (Eng.) Benjamin, S. P. (Dept. Pathology, Cleveland Clinic Foundation, Cleveland, OH 44106) Hawk, W. A. *Lab Invest* 36(3): 330; 1977. (no refs.)

- 77-3409 An Unusual Cerebellar Hamartoma with Dense Core Granules (Meeting Abstract).** (Eng.) Blank, W. F. (Washington Univ. Sch. Medicine, Saint Louis, MO) Nelson, J. S. *Am J Pathol* 86(2): 58a-59a; 1977. (no refs.)

- 77-3410 Electron Microscopic Investigations of Early Stages of Experimental Heart Tumors in Rats.** (Ger.) Holzhausen, H. J. (Pathologisches Institut der Martin-Luther-Universität, DDR-402 Halle, Leninallee 14, E. Germany) Schreiber, D. *Exp Pathol (Jena)* 13(1): 20-31; 1977.

Ultrastructural findings in four early stages of heart neoplasms in the left ventricle of BD-IX rats are reported. The rats received repeated iv injections of methylnitrosourea (20 mg/kg), and they were killed approx 9 mo later. Light microscopy, carried out on semithin heart sections, revealed a parallel array of spindle-shaped tumor cells covered by a layer of endothelium cells. The nuclei of the tumor cells were usually hyperchromatic, although some cells contained pale nuclei. Ultrastructurally, the spindle-shaped cells had irregular nuclei and were loosely arranged. Most cells were surrounded by a basement membrane; thin cell processes and collagen fibers were demonstrable in the extracellular space. The tumors contained characteristic bundles of cell processes as well as single processes, and some large and undifferentiated tumors had thick stumplike processes. Neoplastic cells of an apparently mesenchymal origin were present in the area of the trabecular muscle. The small heart tumors probably developed from neoplastic Schwann cells but with an additional mesenchymal component. Further studies are necessary to clarify both components and their relation to the experimental tumors. (36 refs.)

77-3411 **Neurogenic Origin of Experimental Heart Tumors in the Rat. An Electron Microscopic Study (Meeting Abstract).** (Ger.) Holzhausen, H. J. (Halle/Saale, E. Germany) Schreiber, D. *Zentralbl Allg Pathol* 121(3): 275; 1977. (no refs.)

77-3412 **Ultramicroscopic Observation of Glomic Tumors.** (Fre.) Califano, G (Laboratoire d'Anatomie chirurgicale de la Faculte de Medecine II, Naples, Italy) *C R Soc Biol (Paris)* 170(4): 732-734; 1976.

The gross histology of glomic tumors shows small bands of collagenous fibers separating large nests of cubic or polygonal cells forming a collar around the capillaries. Ultrastructurally, the cells have an abundant cytoplasm rich with fibril and with apparent mitochondria and ribosomes. Nuclear chromatin is abundant, particularly near the peripheral membrane. The cytoplasm has a scarcity of secretory granules. These cells indicate a myoepithelial rather than a chemoreceptor origin for the glomus. (6 refs.)

77-3413 **Isolation and Characterization of a Neuroblastoma Cell Line from Peripheral Blood in a Patient with Disseminated Disease.** (Eng.) Gerson, J. M. (Div. Oncology, Children's Hosp. Philadelphia, 34th & Civic Center Blvd., Philadelphia, PA 19104) Schlesinger, H. R.; Sereni, P.; Moorhead, P. S.; Hummeler, K. *Cancer* 39(6): 2508-2512; 1977.

Upon culture, a sterile, heparinized peripheral blood specimen from a 19-mo-old girl with neuroblastoma yielded a permanent lymphoblastoid cell line. This finding confirms the circulation of neuroblastoma cells in the peripheral blood. (9 refs.)

77-3414 **Disseminated Choroid Plexus Papilloma: An Ultrastructural Study.** (Eng.) Wolfson, W. L. (Div. Neuropathology, Dept. Pathology, Mental Retardation Res. Center, Univ. California Res. Center, Univ. California Center Health Sciences, Los Angeles, CA 90024) Brown, W. J. *Arch Pathol Lab Med* 101(7): 366-368; 1977.

Multiple spinal deposits of choroid plexus papilloma were found in a 16-yr-old boy 5 yr after the removal of a choroid plexus papilloma of the posterior fossa. The fine structure of the metastatic lesions corresponded very closely to that of the normal choroid plexus. A lack of capillary endothelial pores was the only meaningful difference. (15 refs.)

77-3415 **Ultrastructure of a Polysome-Lamellae Complex in a Human Paraganglioma.** (Eng.) Nabarra, B. (Lab. Electron Microscopy, Unit 25, INSERM, Necker

Hosp., 161 Rue de Sevres, 75730 Paris Cedex 15, France) Sonsino, E.; Andrianarison, I. *Am J Pathol* 86(3): 523-532; 1977.

A typical abdominal paraganglioma in a 56-yr-old woman was revealed during surgery and confirmed by histologic observations and topographic and cytologic studies of ultrathin sections. Electron microscopy of the ultrastructure of the polysome-lamellar complex showed an unusual cytoplasmic inclusion appearing as a hollow cylinder, with single or aggregate granules bordering each side of one or several concentric lamellae. In longitudinal section the inclusion was 2-9 microns long; in cross section the external diameter was 0.3-0.9 micron. The one to three lamellae observed were moderately electron-dense and 100-180 Å wide; in most sections they seemed continuous. Layers of 140- to 250-Å particles bordered the lamellae on each side; they were of a ribosomal nature, as suggested by their size and morphologic appearance. The inclusion is described in detail, and hypotheses as to its origin and biological significance are discussed. (26 refs.)

77-3416 **Dense-Core Filaments in Paraganglioma Cells.** (Eng.) Rodriguez Eschandia, E. L. (Departamento de Morfologia, Facultad de Medicina, Universidad Autonoma de Madrid, Madrid, Spain) Nistal, M. *Biol Cell* 28: 83-84; 1977.

An ultrastructural study of a paraganglioma of the glomus jugularis revealed the presence of dense-core filaments composed of approx seven chains of subunits. Six of the subunits made up the wall of the filaments and the seventh was found in the filament core. Clusters of particles interspersed with the filaments were suggestive of alterations in the assembly and turnover of filamentous proteins. (12 refs.)

77-3417 **Mediastinal Paraganglioma Causing Spinal Cord Compression.** (Eng.) Reyes, M. G. (Dept. Neuropathology, Mount Sinai Hosp. Medical Center, Chicago, IL 60608) Fresco, R.; Bruetman, M. E. *J Neurol Neurosurg Psychiatry* 40(3): 276-279; 1977.

The case history of a 24-yr-old woman with mediastinal paraganglioma causing spinal cord compression is presented. A routine chest x-ray disclosed a 2.5-cm mass in the right posterior mediastinum beside the third costovertebral junction. A follow-up radiograph 2 yr later showed the mass to be 5 cm in diameter. A right thoracotomy showed a retropleural, multilobulated paraganglioma that had eroded the head and neck of the second, third, and fourth ribs on the right side. The tumor was excised, except for a small portion lying deep in the third intercostal space in the paravertebral gutter. Four years later the patient was readmitted, presenting with sharp lumbosacral pain and weakness of the legs. X-ray of the thoracic spine demonstrated destruction of the right pedicle of the T4 vertebra. Myelography showed a partial block at the



level of the body of the T4 vertebra. Laminectomy of the T3-T5 vertebrae revealed an epidural tumor that was pushing the spinal cord posteriorly and to the left and was invading the body and pedicles of the T3-T5 vertebrae. The tumor in the epidural space was removed. The patient recovered and, 5 mo later, gave birth to a full-term, normal baby girl. The ultrastructure of this rare neoplasm is described. (10 refs.)

- 77-3418 **Membrane Structures of Human Oligodendroglioma.** (Eng.) Tani, E. (Dept. Neurosurgery, Hyogo Coll. Medicine, Nishinomiya, Hyogo, Japan) Maeda, Y.; Natsume, S.; Ito, Y. *Acta Neuropathol (Berl)* 38(1): 11-19; 1977.

The membrane structures of four human oligodendrogliomas were studied electron microscopically using thin-section and freeze-fracture techniques. The tumor cells were usually ovoid and, occasionally, they possessed short cytoplasmic processes. The nucleus was also round or ovoid. Plasma membrane particles were randomly distributed; the av number of particles was  $1,090/\mu\text{m}^2$  on face A and  $230/\mu\text{m}^2$  on face B. Gap and tight junctions were occasionally visible. The gap junction was often small (about 0.03 micron in size) and composed of a polygonal aggregate of several subunits: particles on face A and pits on face B. Gap particles were 70-80 A in diameter, and the center-to-center distance between particles was about 90 A. Gap pits were approx 30-40 A in diameter, with a center-to-center distance of about 90 A. The gap junction was randomly distributed in the plasma membrane. Gap junctions and ramifying networks of crests or furrows, suggestive of a tight junction, were occasionally seen on the same fracture face of the plasma membrane. The tight junctions in oligodendrogliomas were often continuous, surrounding the cell completely, and they consisted of five strands. (34 refs.)

- 77-3419 **Ganglioside Content and Pattern in Human Gliomas in Culture: Correlation of Morphological Changes with Altered Gangliosides.** (Eng.) Manuelidis, L. (Dept. Pathology, Yale Univ. Sch. Medicine, New Haven, CT 06510) Yu, R. K.; Manuelidis, E. E. *Acta Neuropathol (Berl)* 38(2): 129-135; 1977.

The ganglioside level and pattern of human gliomas in monolayer cultures were examined. These gliomas revealed morphological variations that correlated with several features of ganglioside analysis. Glioblastoma lines TC 178 and TC 501, which had changed morphologically during extended subculture, showed reduced amounts and a simplified pattern of gangliosides, with an almost total loss of the characteristic brain complex gangliosides. In contrast, glioblastoma lines TC 526 and TC 593, as well as the oligodendroglioma line TC 620, had brainlike gangliosides; the cells in these cultures had maintained their characteristic morphology. The possibility that altered ganglioside levels occur in conjunction with

morphological changes after propagation in vitro is discussed. (36 refs.)

- 77-3420 **Genetic Factors in the Pathogenesis of Optic Nerve Glioma (Meeting Abstract).** (Ger.) Gerlach, H. (Halle/Saale, E. Germany) Schneider, M. *Zentralbl Allg Pathol* 121(3): 284-285; 1977. (no refs.)

- 77-3421 **Genetic Control of Brain Tumor Growth (Meeting Abstract).** (Eng.) Albright, L. (Univ. Pittsburgh Sch. Medicine, Pittsburgh, PA) Gill, T. J. *Am J Pathol* 86(2): 22a-23a; 1977. (no refs.)

- 77-3422 **Congenital Ichthyosis and Medulloblastoma.** (Eng.) Walach, N. (Dept. Oncology, Hadassah Univ. Hosp., PO Box 499, Jerusalem, Israel) *Dermatologica* 154(1): 49-52; 1977.

The case is presented of an 18-yr-old man with congenital ichthyosis and medulloblastoma. An etiological relationship was not confirmed. However, if medulloblastoma is viewed as an embryonal tumor, there is a parallelism between the two diseases. (12 refs.)

- 77-3423 **Electron Microscopic Observations of Two Cases of Giant Cell Sarcoma of the Brain In Vivo and In Vitro (Meeting Abstract).** (Ger.) Kubo, O. (Tokyo, Japan) Okino, T.; Kamijo, Y.; Hamada, H.; Himuro, H.; Kitamura, K. *Zentralbl Neurochir* 37(2): 153; 1976. (no refs.)

- 77-3424 **Meningeal Carcinomatosis: Development of an Experimental Model.** (Eng.) Ushio, Y. (Lab. Neuro-Oncology, Dept. Neurology, Memorial Sloan-Kettering Cancer Center, New York, NY) Chernik, N. L.; Posner, J. B.; Shapiro, W. R. *J Neuropathol Exp Neurol* 36(2): 228-244; 1977.

A model of meningeal carcinomatosis was developed in adult female Wistar rats who received various doses of Walker 256 carcinoma by intrathecal injection (under pentobarbital anesthesia). In seven groups of 7-10 rats given  $1 \times 10^4$  to  $2 \times 10^6$  viable tumor cells, the tumor grew in all rats that received  $> 1 \times 10^5$  viable cells. In four groups that received  $1 \times 10^6$  cells, tumor growth was rapid, and death occurred in about 15 days. The histopathological pattern observed was similar to that seen in diffuse leptomeningeal involvement of systemic cancer in human beings. One exception was the prominent epidural invasion by the tumor at the level of the cauda equina. In general, the pathology of the model was highly consistent from animal to animal. Although the animals die in a

short time, with a small difference in mortality from animal to animal, survival is long enough to permit chemotherapy after the tumor has taken and before the animal dies. (16 refs.)

**77-3425 Morphology of the Paraadenomatous Adenohypophysis: A Contribution to the Pathogenesis of Pituitary Adenomas.** (Ger.) Saeger, W. (Pathologisches Institut der Universität Hamburg, Martinistrasse 52, D-2000 Hamburg 20, W. Germany) *Virchows Arch (Pathol Anat)* 372(4): 299-314; 1977.

Ninety-eight biopsies of the paraadenomatous adenohypophysis in acromegaly, galactorrhea, combined acromegaly and galactorrhea, hypothalamic-hypophysial Cushing's disease, Nelson's syndrome, and in nonfunctional adenomas were studied by light microscopy to find evidence for a possible hyperplastic origin of the different types of adenomas. It was hoped that the numerical relations and structures of hypophysial cells might provide significant information. Nodular ACTH cell hyperplasia was frequently found apart from ACTH cell tumors in Cushing's disease and Nelson's syndrome, suggesting that the adenomas in both diseases arise from hyperplasia. During further development, these adenomas seem to become autonomous, since the number of paraadenomatous ACTH cells decreases in larger tumors. Some of the cases with isolated galactorrhea showed hyperplasia of acidophil and chromophobe cells in the paraadenomatous adenohypophysis. Thus, prolactin cell tumors also develop from prolactin cell hyperplasia. Both the adenomas and the nonfunctional tumors in acromegaly seem to grow autonomously from the beginning, since paraadenomatous hyperplasia is rare. (32 refs.)

**77-3426 Pathogenesis of Hypophyseal Adenoma. Investigations on Paraadenomatous Anterior Lobe Tissue (Meeting Abstract).** (Ger.) Saeger, W. (Hamburg, W. Germany) *Zentralbl Allg Pathol* 121(3): 299; 1977. (no refs.)

**77-3427 Ultrastructure and Biochemistry of Thyroid Carcinoma.** (Eng.) Valenta, L. J. (Univ. California at Irvine Medical Center, 101 City Drive S., Orange, CA 92668) Michel-Bechet, M. *Cancer* 40(1): 284-300; 1977.

Thirty-two thyroid tumors (9 benign, 23 malignant) and 12 samples of normal thyroid tissue were examined by light and electron microscopy. Tissue thyroglobulin content was also measured and, in a limited number of cases, enzymatic activities, such as thyroid peroxidase-iodinase, acid protease, and deiodinase, were determined. The presence of significant amounts of 19S, 27S, and 12S thyroglobulin correlated well with the ability of the tumors to accumulate radioiodine. It is suggested that thyroglobulin be used as a marker of poten-

tial function of thyroid carcinoma. Two types of ultrastructural changes were observed in thyroid carcinoma. The first one was interpreted as accompanying the progressive loss of function of thyroid tumors, and it was represented by modifications of highly specialized structures such as rough endoplasmic reticulum, lysosomal dense bodies, and colloid. The second one is suspected to reflect malignant transformation of the follicular cell; namely, the nuclei, mitochondria, and intracytoplasmic inclusions. These changes may have diagnostic value, since they were not observed in benign conditions. (39 refs.)

**77-3428 Certain Aspects of Histogenesis of the B (Ashkenazy) Cells in the Human Thyroid Gland.** (Eng.) Smirnova, E. A. (Lab. Histochemistry and Electron Microscopy, Dept. Pathological Anatomy and Human Tumors, Oncological Res. Center, Acad. Medical Sciences USSR, Moscow 115478, USSR) *Folia Histochem Cytochem (Krakow)* 14(4): 223-226; 1976.

The activity of succinate dehydrogenase and peroxidase was studied in thyroid cancers and adenomas composed of A (follicular cells) or B (Ashkenazy) cells. Histochemical tests for succinate dehydrogenase showed that, regardless of the benign or malignant nature of a tumor or its histological structure, the activity of this enzyme was always low in A-cell tumors and very high in B-cell tumors. The same degrees of activity were seen in normal A and B cells of the thyroid. In tumors composed of both A and B cells, enzyme activity was low in the former and high in the latter. Electron microscopy revealed that the peroxidase activity was localized in the cytoplasmic granules of the A cells and in the mitochondrial cristae of the B cells. These results provide additional proof of the functional and histogenetic differences between A and B cells. (17 refs.)

**77-3429 Karyometric Study of Benign and Malignant Disease of the Thyroid Gland.** (Eng.) Petrova, A. S. (Lab. Clinical Cytology, Oncological Res. Center, Acad. Medical Sciences USSR, Moscow 115478, USSR) Velesvitch, A. V. *Folia Histochem Cytochem (Krakow)* 14(4): 227-232; 1976.

Karyometric measurements were performed on 20 normal thyroids and on the thyroids of patients with thyroid cancer (95), Hashimoto's thyroiditis (20), thyroid adenoma (15), or nontoxic goiters (5). The mean area and mean diameter of the epithelial cell nuclei were calculated for each patient and group. There were significant differences between the mean area of the nuclei of normal thyroid epithelial follicular cells ( $46.35 \mu m^2$ ) and the mean area of those found in benign proliferative processes (goiter, adenoma;  $64.66 \mu m^2$ ), Hashimoto's thyroiditis ( $90.69 \mu m^2$ ), and thyroid cancer ( $116.89 \mu m^2$ ). The shape of the histograms constructed for the cancer patients depended not only on the histologic type of the tu-



mor (follicular, papillary, undifferentiated) but also on cell type (Ashkenazy, follicular, amyloid). The data demonstrate that karyometric measurements can be used in the differential diagnosis of benign and malignant thyroid diseases. (5 refs.)

- 77-3430 **Thyroid Carcinoma as a Late Consequence of Head and Neck Irradiation.** (Dut.) Geerling, J. (No affiliation given) *Ned Tijdschr Geneesk* 120(41): 1754; 1976.

One thousand patients who had undergone x-ray irradiation of the head and neck region for benign diseases during 1939-1962 were investigated for possible changes in the thyroid. The total dose administered was 700 R or higher; 80% of all patients had been irradiated before the age of 8 yr. Nodes were palpable in 141 patients; the scintigraphic findings revealed solitary nodes in 129 cases and multiple nodes in 45. Enlargement of one lobe of the thyroid, or uneven surface of the gland, was observed in 15 patients. Reduced or increased pertechnetate uptake was found in 278 patients. Surgery was performed on 270 patients. Papillary carcinoma was found in 23 cases, follicular carcinoma in three and mixed carcinoma in 34. The other operated patients had benign changes. Nearly 50% of the carcinomas were multicentric, but no distant metastases were found. The latency period was 20-30 yr. (2 refs.)

- 77-3431 **Primary Clear Cell Thyroid Carcinoma with Squamous Features.** (Eng.) Fisher, E. R. (Shadyside Hosp., Pittsburgh, PA 15232) Kim, W. S. *Cancer* 39(6): 2497-2502; 1977.

Electron microscopy of a clear cell thyroid carcinoma with features suggestive of squamous carcinoma indicated that this type of study may be necessary to distinguish a primary tumor from a metastasis. The histogenesis of these tumors is not totally known. (22 refs.)

- 77-3432 **Tumour Metastasis to the Thyroid Gland.** (Eng.) Pillay, S. P. (Dept. Surgery, Univ. Natal, Durban, South Africa) Angorn, I. B.; Baker, L. W. *S Afr Med J* 51(15): 509-512; 1977.

Ten cases (6 women and 4 men aged 40-65 yr) of tumor metastasis to the thyroid gland were reviewed. In six patients, the diagnosis of the primary lesion was obvious from the symptomatology, clinical signs, or results of special investigations. In two patients who presented with disseminated malignancy, renal angiography and prostatic biopsy confirmed the presence of asymptomatic primary tumors in the kidney and prostate, respectively. In the remaining two patients, metastatic tumors of the thyroid gland masqueraded

as a primary neoplasm. Eight patients died within 3 mo of diagnosis. One patient with renal adenocarcinoma was still alive 6 mo after initial diagnosis, but she had developed cannon ball metastases in both lungs. One patient was lost to follow-up. Thyroid involvement implied disseminated malignant disease, but metastatic tumors could simulate a primary thyroid tumor. Needle biopsy of the thyroid gland with the Trucut needle provided histopathological confirmation of the presence of tumor in all patients. Thyroid involvement by metastasis is more common than previously assumed. (12 refs.)

- 77-3433 **Ultrastructural Study of Early Phase of Familial Medullary Carcinoma of Thyroid Gland. Multicentric Parafollicular (C-) Cell Hyperplasia.** (Jpn.) Kakudo, K. (Dept. Pathology, Osaka Univ. Medical Sch., Osaka, Japan) Katayama, S.; Miyaji, T.; Miyauchi, A.; Takai, S.; Kuma, K. *Jpn J Cancer Clin* 23(3): 167-171; 1977.

Two cases of early-phase familial medullary carcinoma of the thyroid gland from two families with multiple endocrine neoplasia syndrome were studied. Ultrastructural examination of the noncancerous follicles of the grossly normal thyroid showed an increased number of C cells on the basement membrane below the follicular cell lining. It was suggested that the increase in C cells was caused by multicentric C-cell hyperplasia and not by intrathyroidal metastasis of medullary carcinoma. (20 refs.)

- 77-3434 **Fatal Thyroid Carcinoma. Anaplastic Transformation of Adenocarcinoma.** (Eng.) Harada, T. (Div. Endocrine Surgery, Kawasaki Medical Sch., Kurashiki, Japan) Ito, K.; Shimaoka, K.; Hosoda, Y.; Yakumaru, K. *Cancer* 39(6): 2588-2596; 1977.

Autopsies of 27 patients with fatal thyroid cancer were performed. Histological studies indicated an apparent transformation from well differentiated cancer to the less differentiated form as part of the natural course of the disease. (27 refs.)

- 77-3435 **Hashimoto's Thyroiditis and its Relationship to Other Thyroid Diseases.** (Eng.) Holmes, H. B. (Dept. Surgery, Medical Univ. South Carolina, Charleston, SC) Kreutner, A.; O'Brien, P. H. *Surg Gynecol Obstet* 144(6): 887-890; 1977.

The autoimmune process that results in Hashimoto's thyroiditis may be premalignant. Of 60 patients with Hashimoto's thyroiditis, 5 had thyroid adenoma (8.3%), 2 had papillary carcinoma (3.3%), and 3 had lymphosarcoma (5.0%). Open biopsy was essential for the accurate diagnosis of Hashimoto's disease and associated carcinomas. (13 refs.)

- 77-3436 **Hodgkin's Disease and Mycosis Fungoides in a Married Couple.** (Eng.) Hazen, P. G. (Div. Dermatology, Univ. Hosps. Cleveland, 2065 Adelbert Road, Cleveland, OH 44106) Michel, B. *Dermatologica* 154(5): 257-260; 1977.

A report is presented of a woman who developed mycosis fungoides 4 yr before her husband developed Hodgkin's disease. In both diseases there is abnormal T-lymphocyte proliferation. Exploratory laparotomy of the husband revealed involvement of the small intestine and the mesenteric and paraaortic lymph nodes, but there was no evidence of systemic involvement in the wife. These two cases concur with other reports on the clustering of lymphoreticular disorders, implying the existence of an infectious agent or a transmissible factor that precipitates the diseases in predisposed patients. The possibility of malignant expression modified by immunocompetence of the respective host must be considered. (14 refs.)

- 77-3437 **Angio-immunoblastic Lymphadenopathy Terminating as Hodgkin's Disease.** (Eng.) Yataganas, X. (First Dept. Internal Medicine, Univ. Athens Medical Sch., Hosp. Vassilevs Pavlos, Athens 609, Greece) Papadimitriou, C.; Pangalis, G.; Loukopoulos, D.; Fessas, P.; Papa-charalampous, N. *Cancer* 39(5): 2183-2189; 1977.

The case history of a 33-yr-old man with generalized lymphadenopathy bearing all physical, laboratory, and histological characteristics of angioimmunoblastic lymphadenopathy with dysproteinemia (AILD) is given. Pathological observations at sequential lymph node biopsies and at autopsy are described. Therapy was without benefit and the patient died 22 mo after initial diagnosis. At autopsy, numerous Hodgkin's cells and Sternberg-Reed cells were identified in the lymph nodes and spleen, in addition to the characteristic cellular polymorphism of AILD. Pleomorphic cellular infiltrates containing an increased number of immunoblasts and some giant cells were found in the portal spaces of the liver. The association of Hodgkin's disease with AILD in this patient indicates that the AILD may have resulted from a reaction to the agent responsible for the development of Hodgkin's disease. (17 refs.)

- 77-3438 **Fatal Histiocytic Lymphoma of the Brain Associated with Hyperimmunoglobulinemia-E and Recurrent Infections.** (Eng.) Bale, J. F. (Div. Clinical Immunology, Dept. Pediatrics, Univ. Utah Coll. Medicine, Salt Lake City, UT 84132) Wilson, J. F.; Hill, H. R. *Cancer* 39(6): 2386-2390; 1977.

The report of a 10-yr-old girl with eczema, asthma, recurrent abscesses, middle ear infections, pneumonia and histiocytic lymphoma is presented. She also had hyperimmunoglobulinemia E. It is suggested that patients with this elevated immunoglobulin may be at risk for the development of unusual neoplasms. (22 refs.)

- 77-3439 **Hodgkin's Disease of the Thymus (Granulomatous Thymoma) and Myasthenia Gravis. A Unique Association.** (Eng.) Null, J. A. (Dept. Pathology, Yale Univ. Sch. Medicine, New Haven, CT 06510) LiVolsi, V. A.; Glenn, W. W. *Am J Clin Pathol* 67(6): 521-525; 1977.

The case history of a 19-yr-old girl with myasthenia gravis and granulomatous thymoma is presented. The patient was well 8 mo after removal of the thymus, indicating that the myasthenia and the Hodgkin's disease were interrelated (12 refs.)

- 77-3440 **Malignant Histiocytosis with Associated "Reticulum Celled Sarcoma."** (Eng.) Marshall, A. H. (Inst. Pathology, London Hosp., Whitechapel El, London, England) Revell, P. A. *J Pathol* 122(1): 9-12; 1977.

A unique case of reticulum cell sarcoma associated with malignant histiocytosis occurred in a 50-yr-old man. A biopsy specimen of a gastric tumor showed destruction of the surface mucosa overlying a poorly differentiated round cell lymphoma. Abnormal giant cell histiocytes were observed in some of the enlarged para-aortic lymph nodes. The findings suggest the existence of a gradient of neoplasia from well-differentiated histiocytes to an anaplastic malignant tumor of uncertain origin. (8 refs.)

- 77-3441 **Ultrastructure of the Spleen in Malignant Histiocytosis.** (Eng.) Heustis, D. G. (Loma Linda Univ. Medical Center, Loma Linda, CA 92354) Bull, B. S.; Hadley, G. G. *Arch Pathol Lab Med* 101(5): 239-242; 1977.

During treatment for a fractured femur that did not heal well, a 40-yr-old man developed severe thrombocytopenia, splenomegaly, leukopenia, thrombophlebitis, and a fever of unknown origin. Bone marrow aspiration yielded a diagnosis of malignant histiocytosis. A splenectomy was performed, and the spleen was examined by light and electron microscopy. The sinuses were greatly dilated and lined by large atypical histiocytic cells, and erythrophagocytosis was prominent. These findings together with histologic examination of the bone marrow and autopsy specimens confirmed the diagnosis of malignant histiocytosis. Because the histiocytes were confined to the sinusoids, the possibility is raised that this condition may represent a rare subvariant, histiocytic sinus reticulosis. (7 refs.)

- 77-3442 **Neoplasms of Probable Histiocytic Nature: A Light (LM) and Electron Microscopic (EM) Study (Meeting Abstract).** (Eng.) Vuletin, J. C. (S.U.N.Y. Downstate Medical Center, Brooklyn, NY) Greco, M. A.; Feiner, H.; Schinella, R. A.; Soloman, M. P. *Lab Invest* 36(3): 359; 1977. (no refs.)



**77-3443 Mitotic Histiocytes and Intranuclear Langerhans Cell Granules in Histiocytosis X. (Eng.)**

Shamoto, M. (Dept. Pathology, Fujita-Gakuen Univ. Sch. Medicine, Kutsukake-cho, Toyoake-City, Aichi-Prefecture, Japan) *Virchows Archiv [Cell Pathol]* 24(1): 87-90; 1977.

Mitotic histiocytes containing Langerhans cell granules were found in specimens of Letterer-Siwe (L-S) disease and Hand-Schuller-Christian (H-S-C) disease. Biopsy specimens of lymph nodes of L-S disease and an sc mass of the right occipital region of H-S-C disease were examined by electron microscopy. Histiocytes in anaphase were observed in the lymph nodes of L-S disease, and Langerhans cell granules were seen near the chromosomes of these nuclei during mitosis. In the H-S-C disease specimens there were Langerhans cell granules, vacuoles, vesicles, and other cytoplasmic material lying free inside a few nuclei. Langerhans cell granules may be trapped in the nucleus during mitosis. The appearance of these granules in the nucleus indicates that the cells containing them undergo mitosis, even though mitotic cells are rarely observed at the ultrastructural level. (9 refs.)

**77-3444 The Benign Lymphoepithelial Lesion of the Salivary Glands. A Case Report. (Eng.)**

Worthington, P. (Dept. Maxillofacial Surgery, Maclor General Hosp., Wrexham LL13 7TD, Clwyd, North Wales) *J Maxillofac Surg* 5(1): 81-86; 1977.

A 61-yr-old man presented with swelling in the left side of the floor of the mouth. At operation, a right submandibular stone was removed intraorally, and the left sublingual gland was biopsied. It resembled a benign lymphoepithelial lesion of the salivary glands. At further surgery, the left sublingual gland, the left submandibular gland, and the lingual nerve were removed. At recall 2 yr later, the patient was found to have further swellings in the floor of the mouth. The right sublingual gland, previously normal clinically, was enlarged and rubbery. Treatment for malignant lymphoma by megavoltage radiotherapy and chemotherapy was instituted, but the patient died 4 yr later. This case supports evidence favoring a definite connection between benign lymphoepithelial lesions and malignant lymphoma. It is suggested that both conditions may be expressions of the same underlying host defect, with mutant cells taking part either in an autoimmune process or in the development of malignant disease of the lymphoid tissue. (28 refs.)

**77-3445 Nuclear and Nucleolar Ultrastructure of Sezary Cells. (Eng.)**

Smetana, K. (Dept. Pharmacology, Baylor Coll. Medicine, Houston, TX 77030) Daskal, Y.; Gyorkey, F.; Gyorkey, P.; Lehane, D. E.; Rudolph, A. H.; Busch, H. *Cancer Res* 37(7): 2036-2042; 1977.

Sezary cells were studied in the peripheral blood (3 patients with mycosis fungoides and 2 patients with Sezary syndrome) and characteristic skin lesions (2 patients with Sezary syn-

drome) by transmission electron microscopy to obtain more information on their nuclear and nucleolar ultrastructure. Sezary cells contain nucleoli with nucleolonemas or ring-shaped nucleoli similar to those of lymphoblasts and mature lymphocytes. "Maturation asynchrony" of the nucleolus and cytoplasm was evident in some cells that contain large numbers of ribosomes and ring-shaped nucleoli and in other cells that contain nucleoli with nucleolonemas and few ribosomes. The maturation asynchrony of the nucleolus and the cytoplasm, the presence of mitochondrionlike inclusion bodies in the nucleus, and fusion of mitochondria with the nucleus in Sezary cells are ultrastructural abnormalities of this neoplastic lymphocytic variant. The presence of the intranuclear mitochondrionlike inclusion body and nuclear rodlets in Sezary cells was an exceptional finding. (21 refs.)

**77-3446 DNA Content of Mycosis Fungoides Cells. (Eng.)**

Hagedorn, M. (Dept. Dermatology, Univ., Hauptstrasse 7, D-7800 Freiburg/Breisgau, W. Germany) Kiefer, G. *Arch Dermatol Res* 258(2): 127-134; 1977.

The DNA content of mycosis infiltrate cells of five patients in the plaque and tumor stages of mycosis fungoides was measured by Feulgen cytophotometry. The infiltrate cells were differentiated cytochemically into histiocytes and atypical lymphoid cells with NaF-sensitive naphthol-AS-D-acetate esterase. Increasing duration of the tumor stage was associated with a larger proportion of tetraploid and octoploid cells, but no aneuploid stem line was demonstrated. The DNA histograms also exhibited a local proliferation of a typical lymphoid cells that was arrested by cytostatic therapy. Comparison with semi-thin-sections of tumor tissue showed that the mycosis fungoides cells are typical lymphoid cells. These DNA measurements were not in conflict with existing cytogenetic evidence for limited aneuploidy in mycosis fungoides. The DNA distribution pattern indicated that mycosis fungoides fits in the group of lymphomas. (30 refs.)

**77-3447 Multiple Myeloma with Cutaneous Involvement. (Eng.)**

Rodriguez, J. M. (Dept. Dermatology, New York Univ. Sch. Medicine, 550 First Ave., New York, NY 10016) Lam, S.; Silber, R. *JAMA* 237(24): 2625-2626; 1977.

A 49-yr-old man with IgA type multiple myeloma subsequently developed cutaneous plasmacytomas. Immunofluorescence failed to demonstrate IgA production in the skin cells. (6 refs.)

**77-3448 Subcorneal Pustulosis and IgA Myelomatosis. (Eng.)**

Cream, J. J. (Charing Cross Hosp., London W6 8RF, England) Grimes, S. M.; Roberts, P. D. *Br Med J* 1(6060): 550; 1977.

The case report is presented of a 61-yr-old man who developed subcorneal pustulosis in connection with IgA myelomatosis; 5 yr earlier, an adenocarcinoma of the sigmoid colon had been removed. Since serum IgA abnormalities have been noted in patients with skin disorders, there may be an etiological connection. (5 refs.)

- 77-3449 **Membranous Structures in Myeloma Cells (Meeting Abstract).** (Eng.) Kondo, K. (Center for Adult Diseases, Osaka, Japan) Yoshitake, J. *J Electron Microsc (Tokyo)* 25(3): 197; 1976. (no refs.)

- 77-3450 **Pathology of 'Non-healing (Midline) Granuloma.'** (Eng.) Michaels, L. (Dept. Pathology, Inst. Laryngology and Otology, Univ. London, London, England) Gregory, M. M. *J Clin Pathol* 30(4): 317-327; 1977.

Biopsy specimens of 10 cases of midline granuloma, or Wegener's granuloma, showing a combination of widespread nasal necrosis and groups of atypical cells were studied. The atypical cells were larger than mononuclear inflammatory cells, and they had nuclei that varied from round to irregularly elongated and bent. The chromatin material was scanty and irregularly distributed. The amount of cytoplasm varied from a little to a lot and often contained phagocytosed basophilic debris. Necrosis was always prominent and usually of the coagulative type. The invasion of adjacent tissue was seen in four cases in which nerve sheaths were invaded, two cases in which skeletal muscle was invaded, and in two cases in which the nasal bone was invaded. Spread to cervical and more distant lymph nodes, spleen, liver, and kidney was observed in several cases. Erythrophagocytic activity in the spleen was seen in three cases and histiocytic infiltration of the bone marrow was observed in two cases. The belief that the malignant neoplasm was a form of lymphoma is discussed. The concept of midline granuloma and its synonyms should be abandoned, and efforts should be concentrated on making an accurate histological diagnosis in obstructive and ulcerating conditions of the nose. (14 refs.)

- 77-3451 **Erythroid Colony Formation by Polycythemia Vera Bone Marrow In Vitro. Dependence on Erythropoietin.** (Eng.) Zanjani, E. D. (Dept. Physiology, Mount Sinai Sch. Medicine, City Univ. New York, New York, NY 10029) Lutton, J. D.; Hoffman, R.; Wasserman, L. R. *J Clin Invest* 59: 841-848; 1977.

The mechanism of endogenous erythroid colony (EEC) formation was investigated in patients with polycythemia vera (PV). In the plasma clot culture system, both normal and PV bone marrow cells responded to erythropoietin (Ep), giving rise to large numbers of colonies of erythroid cells. In PV, but not in normal individuals, the marrow produced EEC in

the absence of exogenous Ep. Exposure, before use in culture, of fetal calf serum and citrated bovine plasma to the immunoglobulin G (IgG) fraction of rabbit anti-Ep serum followed by treatment with goat anti-rabbit IgG resulted in a significant decrease in EEC formation. Addition of anti-Ep serum directly to the culture medium produced similar results. The production of EEC in response to added Ep was inhibited in the presence of anti-Ep. Addition of small doses of highly purified Ep to anti-Ep-treated cultures resulted in the reappearance of a significant number of EEC. The formation of EEC is most probably due to the presence of a population of erythroid precursor cells with altered proliferative characteristics in PV. The results imply that sufficient amounts of Ep were present in the fetal calf serum and citrated bovine plasma to initiate the proliferation and differentiation of these cells. It is concluded that Ep plays an important role in the regulation of erythropoiesis in PV patients as well as in normal individuals. (24 refs.)

- 77-3452 **Radiation-induced Erythroleukemia in the Beagle Dog: A Hematologic Summary of Five Cases.** (Eng.) Tolle, D. V. (Div. Biological and Medical Res., Building 202, Argonne Natl. Lab., 9700 S. Case Ave., Argonne, IL 60439) Fritz, T. E.; Norris, W. P. *Am J Pathol* 87(3): 499-510; 1977.

Eleven cases of myeloproliferative disease occurred in a group of 24 beagle dogs placed in a  $^{60}\text{Co}$   $\gamma$ -ray field at about 13 mo of age and irradiated at an exposure rate of 5 R/22-hr day until death. Of these 11 dogs, 5 were diagnosed as having erythroleukemia. The bone marrow showed marked erythroblastic hyperplasia, with maturation arrest of the erythroid elements, and increased numbers of myeloblasts and promyelocytes. The terminal peripheral blood was characterized by marked anemia and thrombocytopenia, with circulating erythrocytic precursors and abnormal erythrocyte morphology. Splenomegaly and hepatomegaly occurred in 4/5 animals. Extensive leukemic infiltration and proliferation were noted in the spleens and livers of all five animals. The extent of leukemic involvement in other tissues and organs varied in individual dogs. The potential usefulness of the canine system as a model for the study of human erythroleukemia is discussed. (24 refs.)

- 77-3453 **Cytogenetic Studies in Preleukemia Using the G-banding Staining Technique.** (Eng.) Panani, A. (Res. Lab. Professorial Medical Unit Evangelismos Hosp., Athens, Greece) Papayannis, A. G.; Kyrkou, K.; Gardikas, C. *Scand J Haematol* 18(4): 301-308; 1977.

A total of 15 patients (14 men, 1 woman; aged 50-82 yr), characterized as preleukemic, were analyzed cytogenetically utilizing a trypsin-Giemsa banding technique to analyze karyotypes. Nine patients exhibited normal karyotypes, while the other six exhibited abnormalities involving trisomies and deletions. The abnormalities were localized in Group C in all



six cases and in Group A in three cases. Trisomies were identified in chromosomes 8 and 9 and aneuploidy of the C Group chromosomes seemed to be a consistent factor between preleukemia and acute leukemia. These similarities in karyotype between preleukemia and acute leukemia suggest that the malignant process is already present before the disease manifests itself. (26 refs.)

- 77-3454 Immunoblastic Lymphadenopathy Proceeding to Sarcoma (Letter to Editor).** (Eng.) Toth, J. (Dept. Pathology, Peterfy Hosp., 1441 Budapest, Hungary) Garam, T. *Lancet* 1(8002): 102; 1977.

The case history of a 77-yr-old woman with immunoblastic lymphadenopathy that progressed to immunoblastic sarcoma is presented. The lymphadenopathy was diagnosed tentatively, at first, with morphological findings indicating the abnormal immune reaction; sarcoma was not diagnosed until autopsy. (4 refs.)

- 77-3455 Ultrastructural Characteristics of the Lymph Node Cells in Lymphosarcoma and Reticulum Cell Sarcoma.** (Rus.) Chinchaladze, T. V. (Lab. Cytomorphology and Leukemia Cytogenetics, Inst. Hematology and Blood Transfusion, Georgian SSR Ministry of Public Health, Tbilisi, USSR) Raikhlin, N. T.; Filipova, N. A.; Bukhvalov, I. B.; Probatova, N. A. *Arkhl Patol* 39(1): 38-43; 1977.

Lymph node specimens from four patients with lymphosarcoma and three patients with reticulosarcoma were analyzed electron microscopically. Three types of cells showing different degrees of maturity were found in the lymphosarcoma tissue. Reticulosarcoma cells retained some of the ultrastructural characteristics of normal reticulocytes (irregular shape, plasma membrane processes, vacuoles in the cytoplasm). The ultrastructural features of these tumor cells might be of value in distinguishing lymphosarcomas from reticulosarcomas. (16 refs.)

- 77-3456 Decrease in Adherence of Cultured Leukemia and Lymphoma Cells to Glucocorticoid Treated Endothelial Cells (Meeting Abstract).** (Eng.) Maca, R. (Univ. Iowa Coll. Medicine, Iowa City, IA 52242) Hakes, A.; Fry, G. *Proc Am Assoc Cancer Res* 18: 152; 1977. (no refs.)

- 77-3457 Endoplasmic Reticulum-associated Structures in Lymphocytes from Patients with Chronic Lymphocytic Leukemia.** (Eng.) Stefani, S. (Therapeutic Radiology Service, 114B, Veterans Admin. Hosp., Hines, IL 60141) Chandra, S.; Schrek, R.; Tonaki, H.; Knospe, W. H. *Blood* 50(1): 125-139; 1977.

The fresh peripheral blood lymphocytes of 61 patients with chronic lymphocytic leukemia (CLL), 28 patients with other lymphoproliferative neoplasms (Hodgkin's disease, lymphosarcoma, and multiple myeloma) and 64 noncancerous human subjects were examined by electron microscopy. Lymphocytes from 13 CLL patients contained three endoplasmic reticulum (ER)-associated structures. Six of these patients had some drug treatment either for CLL or for some other malignancy, but seven had no cancer chemotherapy prior to the first blood sample collection. These structures were either fibrillar, crystalline, or granulofilamentous. Except for the crystalline structure that occurred in 80%-90% of the lymphocytes of one CLL patient, the other ER-associated structures were observed infrequently. Lymphocytes from the noncancerous subjects and from the patients with other lymphoproliferative neoplasms did not contain these structures. The occurrence of these structures in untreated patients and their persistence in the lymphocytes of seven patients who provided two or more samples during a 9-yr period suggested a physiologic defect in the leukemic lymphocytes and supported the hypothesis of a clonal origin of these lymphocytes. The presence of the ER-associated structures in the lymphocytes of a small percentage of CLL patients was not considered pathognomonic of the disease. The structures were related to neither the chemotherapy administered to the patients nor to their lymphocyte count during the course of the disease. (28 refs.)

- 77-3458 Subacute Myelocytic Leukemia Associated with the Philadelphia Chromosome and Supplementary Translocation: 9-12.** (Eng.) Dor, J. F. (Clinique Medicale B, CHU Nord, Bd Pierre Dramart, 13326 Marseille Cedex 3, France) Mattei, J. F.; Mattei, M. G.; Mongin, M.; Giraud, F. *Biomedicine* 27: 131-134; 1977.

A 49-yr-old woman with subacute myelocytic leukemia presented with severe clinical symptoms. Cytogenetic findings showed the Philadelphia chromosome, t(9-22), and a second translocation between chromosome 12 and chromosome 9, t(9-12). This second translocation represents a supplementary cytogenetic argument for the isolation of subacute myeloid leukemia with Philadelphia chromosome with the classification chronic myeloid leukemia. (14 refs.)

- 77-3459 The Consequences of the Philadelphia Chromosome Rearrangement in Chronic Myeloid Leukemia (Letter to Editor).** (Eng.) Hecht, F. (Dept. Pediatrics, Child Development and Rehabilitation Center, Univ. Oregon Health Sciences Center, Portland, OR 97201) McCaw, B. K. *Hum Genet* 36(1): 127-128; 1977.

Based on evidence from a man and two of his children, who had an unusually short chromosome No. 22 due to a translocation with chromosome No. 11, a previous investigator concluded that chromosome band 22q12 is concerned with cell

proliferation in chronic myeloid leukemia (CML). The present authors refute this conclusion for the following reasons: the Philadelphia rearrangement found in most patients with CML is a somatic cell mutation involving erythromyeloid cells. The Philadelphia translocation between chromosome No. 22 and other autosomes occurs not only with chromosome No. 9, but also with Nos. 2, 10, 13, 17, 19, 21, or 22. However, no case of CML with an 11;22 rearrangement has ever been reported. In addition, there is no evidence that a locus is rendered inoperative or operative by a Philadelphia translocation or that only a single locus is involved. (2 refs.)

- 77-3460 **Examination of Haptoglobin Phenotypes in the Blood Serum of Patients with Chronic Lymphocytic Leukemia and Their Relatives.** (Rus.) Babaian, R. A. (Central Inst. Hematology and Blood Transfusion, Acad. Medical Sciences USSR, Moscow, USSR) Belostotskii, V. M.; Tokarev, Iu. N. *Probl Gematol Pereliv Krovi* 22(4): 22-24; 1977.

The association between chronic lymphocytic leukemia (CLL) and serum haptoglobin (Hp) type was studied. The distribution of Hp phenotypes in 25 patients with CLL was compared to that in the normal direct blood relatives of the patients and in a matching population (donors). There was a higher frequency of the Hp<sub>1-1</sub> phenotype in the CLL patients (7/25) than in their siblings (6/40) or in the donors (14/100). To what extent this association has anything to do with the genetic predisposition of a person with the Hp<sub>1-1</sub> phenotype to CLL and whether relatives constitute an increased-risk group remains unclear. (9 refs.)

- 77-3461 **Human B Cell, T Cell and Null Cell Leukaemic Cell Lines Derived from Acute Lymphoblastic Leukaemias.** (Eng.) Miyoshi, I. (Dept. Medicine, Okayama Univ. Medical Sch., Okayama 700, Japan) Hiraki, S.; Tsubota, T.; Kubonishi, I.; Matsuda, Y.; Nakayama, T.; Kishimoto, H.; Kimura, I.; Masuji, H. *Nature* 267(5614): 843-844; 1977.

B-cell, T-cell, and null-cell lines newly established from three patients with acute lymphoblastic leukemia (ALL) are described. The B-cell line (BALL-1) was derived from the peripheral blood of a 75-yr-old man with ALL. The peripheral WBC count at the initiation of culture was  $25 \times 10^4/\text{cm}^3$ , with 83% lymphoblasts. The peripheral lymphoblasts and BALL-1 cells were surface immunoglobulin (Ig)-positive for the two heavy-chain classes IgM and IgA. Cells from both sources contained 36%-60% Fc fragment of IgG (EA) and complement (EAC) rosetting cells, but neither of them formed rosettes with sheep erythrocytes (E). The T-cell line (TALL-1) was established from the bone marrow of a 28-yr-old man who developed the terminal leukemic phase of lymphosarcoma. Most marrow lymphoblasts and TALL-1 cells

formed E rosettes, but neither of them bore surface Ig. The null-cell line (NALL-1) was obtained from a 14-yr-old boy with ALL. Neither peripheral lymphoblasts nor NALL-1 cells demonstrated E-rosette formation or surface Ig. Receptors for EAC but not for EA were found on a small percentage of the NALL-1 cells. Karyotypic analysis showed that BALL-1 cells had a pseudodiploid mode with four characteristic markers, TALL-1 cells had a hypertetraploid mode with 95-101 chromosomes, and NALL-1 cells had a hypodiploid mode with 43 chromosomes. Indirect membrane immunofluorescence showed that only TALL-1 cells were fluorescent with antithymocyte serum, indicating that BALL-1 and NALL-1 cells lacked thymus-related antigen. The use of these cell lines should contribute to a further understanding of human lymphocyte subpopulations in normal and leukemic states. (14 refs.)

- 77-3462 **Establishment in Continuous Culture and Characterization of Cell Surface Markers, and Other Immunologic and Virologic Properties of Lymphoblastoid Cells Derived from Patients with Different Types of Leukemia.** (Eng.) Goldblum, N. (Chanock Center for Virology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel) *Isr J Med Sci* 13(7): 725-730; 1977.

The properties of 63 lymphoblastoid cell lines from patients with various types of lymphoma are described. All but a few of these continuous cell lines exhibit characteristics typical of lymphoblastoid cell lines: they are pleomorphic and contain "hand-mirror"-shaped forms; they are all of B-cell origin; they carry the Epstein-Barr virus (EBV) genome; and they exhibit a sharp decrease in cap-forming ability with fluorescent concanavalin A (Con A) upon passage in culture. One cell line, DG-75, derived from a "Burkitt-like" malignant lymphoma, is characterized by absence of the EBV genome and receptors for EBV. DG-75 resembles the African (EBV-positive) Burkitt's lymphoma cell lines in the following properties: gross morphology, B-type cell properties seen by scanning electron microscopy, and abnormality in chromosome 14. It differs from these cell lines in growth characteristics, high cap-forming ability, low agglutinability with Con A, and the absence of rosette formation (both erythrocyte and erythrocyte-antibody-complement). It is suggested that the geographic designations of African, American, and other Burkitt types be abandoned in favor of classification of Burkitt's lymphoma as being either EBV-positive or EBV-negative (as DG-75). (29 refs.)

- 77-3463 **Hairy Cell Leukemia in the Spleen.** (Fre.) Diebold, J. (Service Central (Jacques-Delarue) d'Anatomie et de Cytologie pathologiques, Hotel-Dieu, place du Parvis Notre-Dame, F-75181 Paris Cedex 04, France) Chomette, G.; Reynes, M.; Tricot, G. *Virchows Arch (Pathol Anat)* 372(4): 299-314; 1977.



Spleens from 15 patients with hairy cell leukemia were studied by immunofluorescence (7 cases), electron microscopy (5 cases), and the usual histologic methods. The findings are: enlargement of the spleen (wt > 400 g); diffuse red pulp infiltration by ambiguous cells with regular separation of nuclei by clear cytoplasm; hairy aspects of the cytoplasmic membrane, as observed on semithin and ultrathin sections; and the presence of cytoplasmic inclusion bodies (polysome lamellae complex). Cellular infiltration was accompanied by some vascular modifications, such as pseudoangiomatosis lesions and nodular formations resembling splenic tumors. Hypertrophy of the splenic macrophages with erythrophagocytosis and siderosis was also observed. These two phenomena partly explain the anemia. (28 refs.)

**77-3464 In Vivo and In Vitro Stem Cell Kinetics in Normal and Erythroleukemic Mice (Meeting Abstract).** (Eng.) Koltun, L. A. (New York Univ., New York, NY 10003) *Diss Abstr Int [B]* 37(9): 4277; 1977. (no refs.)

**77-3465 Surface Morphology of Leukemic Cells: Application of Scanning Electron Microscopy to the Study of Human Leukemias.** (Eng.) Polliack, A. (Dept. Hematology, Hadassah Univ. Hosp., Jerusalem, Israel) *Isr J Med Sci* 13(7): 701-709; 1977.

Scanning electron microscopy studies of the surface architecture of leukemic cells from 184 cases of acute and chronic leukemia are summarized. In general, leukemic cells had surface features similar to those of their normal counterparts. However, cells from patients with lymphoblastic leukemias could be distinguished from those of patients with myeloid and monocytic leukemias on the basis of surface architecture. Leukemic monocytes, myeloblasts, promyelocytes, and maturing myeloid bodies exhibited ridgelike profiles and ruffles, but leukemic lymphocytes and lymphoblasts had varying numbers of microvilli. Cells from patients with lymphoblastic leukemia consistently displayed few surface microvilli, but leukemic lymphocytes from patients with chronic lymphocytic leukemia and other lymphoproliferative disorders generally had more villous surfaces. (23 refs.)

**77-3466 Electron Microscopic Observations of Cell Coat of Childhood Leukemic Cells Using Concanavalin A-Horseradish Peroxidase Method.** (Eng.) Eguchi, M. (Dept. Pediatrics, Faculty Medicine, Shinshu Univ., Matsumoto, Japan) Komiyama, A.; Hanamura, K.; Tsukada, M.; Akabane, T. *Acta Haematol Jpn* 40(1): 33-41; 1977.

The leukemic cell coat was studied by electron microscopy after it was stained by the concanavalin A-horseradish perox-

idase (Con A-HRP) method. Bone marrow specimens were obtained from 15 children with acute leukemia [6 acute lymphoblastic leukemia (ALL), 3 acute myeloblastic leukemia (AML), and 6 acute blastic leukemia (ABL)], 1 child with chronic myelocytic leukemia (CML), and 5 normal children. The thickness of the cell coat was measured quantitatively with a planimeter and a curvimeter. The thicknesses of the cell coat reaction products of leukemic cells from AML, ALL, and ABL were 369, 447, and 459 Å, respectively. These values were significantly greater than those of normal neutrophils (278-303 Å). The thickness of the reaction product of the CML WBC was not significantly different from normal neutrophils, and there was no significant difference between the thickness of the reaction product of leukemic cells from ALL and ABL and normal lymphocytes. The significance of the different thicknesses among leukemic cells or normal WBC remains obscure. They may be due to differences in the malignant cell coats, differences among cell types, or to cell maturity or atypism. (20 refs.)

**77-3467 Neoplastic Macrophages Grown from Human Leukaemic Monocytes.** (Eng.) Balkwill, F. R. (ICRF Labs., Lincoln's Inn Fields, London WC1, England) Franks, C. R.; Oliver, R. T.; Spector, W. G. *J Pathol* 122(1): 13-26; 1977.

Sixty of the blood specimens from 70 untreated patients with acute myelogenous leukemia (AML) produced cells that adhered to plastic flasks when the cultures were examined on the third or fourth day. Ten specimens that produced > 50 adherent cells per high power field were investigated further. Cultures of these cells were trypsin-resistant, phagocytic, peroxidase-negative, and phosphatase-positive after 4 days, positive for binding of antimacrophage serum, and they showed Fc and C<sub>3</sub> receptors. Long-term culture of AML WBC showed a steady increase in the number of macrophages from 3 to 14 days, followed by a decline. The increase was partly due to the capacity of the macrophages to divide in vitro. Ultrastructurally, the macrophages showed many abnormal features characteristic of neoplastic cells. Sc injections of AML cells from 6/10 patients into immune-deprived female CBA mice caused solid tumors with a uniform cell pattern and a malignant reticulum cell appearance. Some human lymph node tumors may be composed of similar populations of macrophages and arise from similar precursors. (20 refs.)

**77-3468 An Autopsy Case of Aplastic Anemia-PNH Syndrome Terminated in Acute Granulocytic Leukemia.** (Eng.) Hiroshige, Y. (Third Dept. Internal Medicine, Yamaguchi Univ. Sch. Medicine, Ube, Japan) Matsumoto, N.; Harima, K.; Miwa, S.; Kamei, T.; Ishihara, T. *Acta Haematol Jpn* 40(1): 16-23; 1977.

The case of a 28-yr-old woman with paroxysmal nocturnal hemoglobinuria (PNH)-aplastic anemia syndrome, which terminated in acute granulocytic leukemia 3 yr after the diagnosis of PNH, is presented along with autopsy findings. In a review of the literature, 2/4 cases of PNH that terminated in acute granulocytic leukemia also showed monocytosis in the peripheral blood as a preleukemic blood picture. Bone marrow abnormalities in these cases were characterized by erythroid hyperplasia. PNH and bone marrow aplasia might be equally responsible for the development of acute leukemia in this case. (16 refs.)

- 77-3469 **Fanconi Anemia Leading to Acute Myelomonocytic Leukemia.** Cytogenetic Studies. (Eng.) Bourgeois, C. A. (Paediatric Res. Unit, Prince Philip Lab., Guy's Hosp. Medical Sch., London, England) Hill, F. G. *Cancer* 39(3): 1163-1167; 1977.

It is speculated that therapy with vincristine and oxymetholone could have played a causative role in the development of myelomonocytic leukemia in a child with Fanconi's anemia. A bone marrow smear revealed a marker chromosome similar to one seen previously in a patient who had undergone a similar evolutionary pattern. (24 refs.)

- 77-3470 **Monomyelocytic Leukemia in an Untreated Case of Waldenstrom Macroglobulinemia.** (Eng.) Salberg, D. (Dept. Immunology, Univ. Michigan Sch. Medicine, Ann Arbor, MI 48104) Kurtides, E. S.; McKeever, W. P. *Arch Intern Med* 137(4): 514-516; 1977.

A 68-yr-old woman with untreated Waldenstrom macroglobulinemia developed acute monomyelocytic leukemia 4 yr later. Since she had not received chemotherapy or radiation, spontaneous transformation was evident. (23 refs.)

- 77-3471 **Granulopoietic Studies in Acute Lymphocytic Leukemia of Children.** (Eng.) Mangalik, A. (Dept. Pediatrics, 4200 East Ninth Ave., Denver, CO 80220) Robinson, W. A.; Holton, C. P. *Blut* 34(2): 77-88; 1977.

Studies were carried out on the levels of serum and urine colony stimulating activity (CSA) and peripheral blood and bone marrow colony forming cell numbers in children with acute lymphocytic leukemia (ALL) suggest that serum and urine levels of colony stimulating factor are reduced during the initial or relapse phase of the disease compared to levels found during remission. The number of bone marrow colony forming cells was reduced in relapse or before treatment and elevated during remission while the number of peripheral blood colony forming cells was increased during relapse or before treatment and normal during remission. It was also shown that mixing of serum or leukemic cells with

normal human bone marrow cells inhibits colony formation ( $p < 0.005$ ). This and other studies suggest that the suppression of normal hematopoiesis in ALL may result from factors produced by leukemic cells and found in the serum. (23 refs.)

- 77-3472 **Studies on In Vitro Differentiation of Acute Myelogenous Leukaemia Cells (Meeting Abstract).** (Eng.) Balkwill, F. R. (I.C.R.F. Dept. Medical Oncology, St. Bartholomew's Hosp., London EC1A 7BE, England) Oliver, R. T. *Br J Cancer* 35(2): 246-247; 1977. (no refs.)

- 77-3473 **Electron Microscopy in Hand-Mirror-Cell Leukaemia (Letter to Editor).** (Eng.) Schumacher, H. R. (Div. Hematopathology and Electron Microscopy, Dept. Lab. Medicine, Natl. Naval Medical Center, Bethesda, MD 20014) Robinson, J. J.; Creegan, W. J.; Pitts, L. L.; Stass, S. A. *Lancet* 1(8023): 1214-1215; 1977.

Electron micrographs of blast cells from a patient with hand-mirror-cell leukemia revealed a large number of undamaged mitochondria. As in a previously reported case of acute myeloblastic leukemia, the mitochondria seemed to be associated with prolonged survival. (5 refs.)

- 77-3474 **Cytodifferentiation in Human Acute Myeloblastic Leukaemia (Meeting Abstract).** (Eng.) Lan, S. (Ontario Cancer Inst., Toronto, Canada) McCulloch, E. A.; Till, J. E. *Br J Cancer* 35(2): 246; 1977. (no refs.)

- 77-3475 **Physiological and Environmental Aspects of L 1210 Murine Leukemia (Meeting Abstract).** (Eng.) Szilagyi, J. E. (Ohio State Univ., Columbus, OH 43210) *Diss Abstr Int [B]* 37(8): 3803; 1977. (no refs.)

- 77-3476 **A Rat Myeloid Leukemia Model of the Human Disease. Hematological Characteristics of N-Nitrosobutylurea-induced W/Fu Rat Leukemia L1504 and L1005.** (Eng.) Okada, K. (Dept. Internal Medicine, Res. Inst. Nuclear Medicine and Biology, Hiroshima Univ., Hiroshima, Japan) Teratani, M.; Takahashi, A.; Uchino, H.; Kajihara, H.; Ohkita, T. *Acta Haematol Jpn* 40(1): 72-86; 1977.

Two myeloid leukemias of W/Fu rats, both of which were induced by the po administration of N-nitrosobutylurea (BNU), were investigated. L1504 was induced in a male rat by administration of BNU (5 mg/day) in drinking water for 6 mo starting at 8 wk of age. It was transplanted iv or ip into 6- to 12-wk-old male rats. L1005 was induced similarly in a



female rat and transplanted into 6- to 12-wk-old females. L1504 showed rapid growth and a negative peroxidase reaction. Its transplantability was 100%, with a narrow range of survival. Morphologically L1504 consisted uniformly of blast cells in the peripheral blood. L1005 was relatively slow-growing, with a positive peroxidase reaction, and it resembled chloroleukemia in gross pathology. Its transplantability was 97%-100% after injection of  $> 10^3$  cells. L1005 consisted of leukemic myeloblasts and young promyelocytes with a tendency toward maturation. There was an inverse correlation between the number of transplanted cells and survival time for both tumors. The hematological characteristics of both L1504 and L1005, such as high WBC counts, abnormal differentials, low platelets and Hb in the peripheral blood, and bone marrow pictures, correlated well with the progression of leukemia, suggesting that they would be suitable models for the study and treatment of human myeloid leukemias. (35 refs.)

- 77-3477 **Variability in Differentiation Stages Observed in a Rat Myeloid Leukemia and Its Relation to Human Myeloid Leukemia.** (Eng.) Okada, K. (Dept. Internal Medicine, Res. Inst. Nuclear Medicine and Biology, Hiroshima Univ., Hiroshima, Japan) Teratani, M.; Takahashi, A.; Mikami, M.; Okita, H.; Kamada, N.; Ucinio, H.; Ohkita, T. *Acta Haematol Jpn* 40(1): 87-106; 1977.

Changes in the biological characteristics of leukemic cells during successive ia-iv transplantations of an acute myeloid leukemia (L1005) induced by N-nitrosobutylurea (BNU) were studied in W/Fu-rats. Ia-iv transplantations of L1005 were done successively from the fifth generation using  $10^5$  cells. Leukemic cells with a negative peroxidase reaction appeared spontaneously at the eighth generation (L1005PO-). New variants appeared repeatedly after the eighth generation. The peroxidase-positive group showed eye discharge, postlimb paralysis, green, yellow-green, yellow-red, or red-white marrows, azure granules in the cytoplasm, and a tendency toward maturation; it was designated the differentiated type. The peroxidase-negative group displayed a red marrow and no clinical signs or azure granules, but it had an apparent leukemic hiatus; it was designated the undifferentiated type. It is not clear whether the mechanisms of conversion from the differentiated to the undifferentiated type and vice versa result from clonal selection of preexisting cells or the appearance of new mutants. It is suggested, however, that a polyclonal situation might exist in human acute myeloid leukemias and that relapses might depend partly on the remaining chemo- and immunoresistant cells after chemo- and immunotherapy. (29 refs.)

- 77-3478 **Acute Myelomonocytic Leukemia in a Patient with Macroglobulinemia and Malignant Lymphoma.** (Eng.) Ligorsky, R. D. (Suite 23-2020 W. Indian School Road, Phoenix, AZ 85015) Axelrod, A. R.; Mandell,

G. H.; Palutke, M.; Prasad, A. S. *Cancer* 39(3): 1156-1162 1977.

Acute myelomonocytic leukemia occurred in a 68-yr-old man with malignant lymphoma and macroglobulinemia. Fluorescent staining demonstrated the macroglobulin in plasma and leukemia cells. Transmission electron microscopy suggested that the plasma cells produced paraproteins that were engulfed by the leukemia cells. The sequence of events ruled out the possibility that the myelomonocytic leukemia developed after therapy with cytotoxic agents. The case is a rare example of the coexistence of malignant lymphoma, macroglobulinemia, and acute myelomonocytic leukemia. (23 refs.)

- 77-3479 **Features Specific to Karyotype of the Lymphatic System Neoplasm.** (Rus.) Fleishman, E. V. (Lab. Cytogenetics, Oncological Scientific Center, Acad. Medical Sciences USSR, Moscow, USSR) Prigozhina, E. L.; Kruglova, G. V.; Volkova, M. A. *Probl Gematol Pereliv Krovi* 22(4): 16-22; 1977.

The karyotypes of 8 patients with chronic lymphocytic leukemia (CLL), 11 patients with lymphosarcoma, and 3 patients with reticulosarcoma were analyzed. An aneuploid karyotype was observed in 3/8 patients with CLL: two patients had deletion of the long arm of chromosome 11 (in 1 the deleted segment was translocated to the long arm of chromosome 17 and in the other, to the short arm of chromosome 2), and the third patient had the marker 14q+ chromosome. A normal diploid karyotype was found in 5/11 patients with lymphosarcoma: 1 patient had ring chromosome 14, 2 patients had similar marker telocentric chromosomes that substituted for the D-group chromosome (in both cases the terminal region of the telocentric arose from the long arm of chromosome 11), and 3 patients had trisomy 3 and 18. One patient with reticulosarcoma had a normal chromosome complement, but two had a hyperdiploid karyotype (1 had rearrangements in the long arm of chromosome 11 and trisomy 18, and 1 had multiple rearrangements involving chromosomes 3, 11, and 14). (23 refs.)

- 77-3480 **Tissue Culture Studies of Hodgkin's Disease (Meeting Abstract).** (Eng.) Long, J. C. (Dept. Pathology, Harvard Medical Sch., Boston, MA 02114) *Lab Invest* 36(3): 346; 1977. (no refs.)

- 77-3481 **Tissue Culture Studies in Hodgkin's Disease: Morphologic, Cytogenetic, Cell Surface, and Enzymatic Properties of Cultures Derived from Splenic Tumors.** (Eng.) Long, J. C. (Immunopathology Lab., Box 9, Massachusetts General Hosp., Boston, MA 02114) Zamecnik, P. A.; Aisenberg, A. C.; Atkins, L. *J Exp Med* 145(6): 1484-1500; 1977.

Monolayer and suspension cell cultures from Hodgkin's tumors in the spleen were examined microscopically and by cytogenetics, tested for lymphocyte and monocyte cell surface properties, and assayed for enzymes histochemically and spectrophotometrically. The monolayer cultures were composed of rapidly proliferating round and polygonal cells that were capable of indefinite propagation in vitro. Abnormal aneuploid chromosomes were present in monolayers passaged 18-20 times and in established cell lines carried in culture > 3 yr and passaged > 200 times. Cells from the monolayers contained lysozyme, fluoride-resistant  $\alpha$ -naphthol acetate esterase, acid and alkaline phosphatase, and chymotrypsinlike activity. The monolayers did not exhibit specific cell surface markers or phagocytosis. Suspension cultures were composed of cells with aneuploid karyotypes and similar enzymes. The cells had surface receptors for complement and IgGfC (crystallizable fragment). The cells lacked surface or cytoplasmic immunoglobulin and did not form E rosettes, react with antithymocyte serum, or exhibit phagocytosis. These findings indicate that the cells of the Hodgkin's disease monolayer and suspension cultures were neoplastic monocytes rather than lymphocytes or fibroblasts. The presence of aneuploid karyotypes is consistent with derivation from a malignant Hodgkin's cell. (34 refs.)

- 77-3482 **HLA in Familial Hodgkin's Disease. Results and a New Hypothesis.** (Eng.) Marshall, W. H. (Immunology Res. Group, Faculty Medicine, Memorial Univ. Newfoundland, St. John's, Newfoundland A1C 5S7, Canada) Barnard, J. M.; Buehler, S. K.; Crumley, J.; Larsen, B. *Int J Cancer* 19(4): 450-455; 1977.

A Newfoundland family with seven cases of Hodgkin's disease (HD) was investigated by HLA typing. Over 600 people in the immediate population were HLA-typed, and clear haplotypes were obtained in about 95%. No simple relationship between HLA haplotype and HD could be found in this familial aggregate. Investigation of the antigen frequencies in the entire population showed that HLA B18 increased progressively in incidence from 0.08 to 0.4 in successive groups of individuals, each more closely related to the HD cases. The highest incidence of B18 was found in the community with the highest incidence of HD. It was suggested that the important HLA association is at a population level rather than at the level of the individual patient. A hypothesis based on the concept of the healthy carrier is presented. A B18-linked complement deficiency could be responsible for such a carrier state. (6 refs.)

- 77-3483 **Lymphoproliferative Disorders in Organ Transplant Recipients (Meeting Abstract).** (Eng.) Hertel, B. F. (Dept. Lab. Medicine, Univ. Minnesota Medical Sch., Minneapolis, MN 55455) Rosai, J.; Dehner, L. P.; Simmons, R. L. *Lab Invest* 36(3): 340; 1977. (no refs.)

- 77-3484 **Development of Malignant Double Gammopathies from Benign Monoclonal Gammopathies (BMG) (Meeting Abstract).** (Eng.) Otto, S. (Natl. Oncological Inst., Budapest, Hungary) Eckhardt, S.; Borzsonyi, M.; Gergely, J. *Scand J Immunol* 6(6/7): 734; 1977. (no refs.)

- 77-3485 **Immunological Disorders and Malignancies in Five Young Brothers.** (Eng.) Putilo, D. T. (Dept. Pathology, Univ. Massachusetts Medical Sch., 55 Lake Ave. N., Worcester, MA 01605) Riordan, J. A.; De-florio, D.; Yang, J. P.; Sun, P.; Vawter, G. *Arch Dis Child* 52(4): 310-313; 1977.

The case histories of five brothers, ranging in age from 6 to 18 yr, who experienced immunological and neoplastic disorders during an 8-yr interval are presented. Two of the brothers succumbed to Grade IV astrocytoma (glioblastoma multiforme) of the brain and another brother died of metastatic carcinoma involving the rectum, liver, spleen, lymph nodes, and lungs. One of the surviving brothers had histiocytic lymphoma and the other surviving brother had idiopathic thrombocytopenia purpura. The mother of the boys was healthy, but her twin sister died in utero of severe malformations. The father of the boys had bronchiectasis 20 yr previously. The parents and one boy were karyotyped, but no abnormalities were observed. It was suggested that the boys were rendered susceptible to oncogenesis by an intrinsic cellular defect inherited from their mother. (14 refs.)

- 77-3486 **Cancer in a Familial IgA Deficiency Patient: Abnormal Chromosomes and B Lymphocytes.** (Eng.) Goh, K. O. (Monroe Community Hosp., 435 E. Henrietta Rd., Rochester, NY 14603) Reddy, M. M.; Webb, D. R. *Oncology* 33(5-6): 237-240; 1976.

The case history of a patient with familial immunoglobulin A (IgA) deficiency and nonspecific chromosomal abnormalities who developed two primary cancers is presented. The patient was a 45-yr-old woman with a long history of chronic lung disease and an isolated IgA deficiency. Three months prior to her final admission, the patient underwent a transverse colostomy to relieve a bowel obstruction caused by adenocarcinoma of the rectum. Following this operation she developed a clear cone lesion in the lung. Sputum cytology examination revealed malignant squamous cells. X-rays showed two small soft tissue densities, one in the left upper apical lobe and the other behind the inferior vena cava. The patient refused further exploration and died 3 wk later. No postmortem examination was done. Cytogenetic studies in peripheral blood lymphocyte cultures showed a nonconsistent pseudodiploid chromosomal pattern. An E(17-18) chromosome with a deleted short arm was seen in two metaphases. In several other metaphases this chromosome was abnormally large. The patient also showed a deficiency in the number of B lymphocytes in the peripheral blood. The ab-



sence of IgA, the low number of peripheral B lymphocytes, and the chromosomal abnormality may have played a role in the susceptibility of this patient to cancer. (23 refs.)

- 77-3487 **Alzheimer's Disease, Trisomy 21, and Myeloproliferative Disorders: Associations Suggesting a Genetic Diathesis.** (Eng.) Heston, L. L. (Dept. Psychiatry, Univ. Minnesota, Minneapolis, MN 55455) *Science* 196(4287): 322-323; 1977.

Of 857 first and second degree relatives of 30 probands with Alzheimer's disease, 22 had Alzheimer's disease, 6 had trisomy 21, and 13 had myeloproliferative disorders. It is suggested that this high incidence resulted from a genetic defect which caused a predisposition to these afflictions and was manifested in the pathological microtubule development found in all three disorders. (15 refs.)

- 77-3488 **Double Primary Cancers in 2 Young Sibs, Leukemia in Another, and Dextrocardia in a Fourth.** (Eng.) Li, F. P. (35 Binney St., Boston, MA 02115) McIntosh, S.; Peng-Whang, J. *Cancer* 39(6): 2633-2636; 1977.

The case histories are presented of three brothers, one with glioblastoma and non-Hodgkin's lymphoma, another with a brain tumor and acute leukemia, and a third with myelogenous leukemia. A fourth had cyanotic congenital heart disease. The various etiological factors involved are discussed. No diagnostic laboratory markers of a familial syndrome with susceptibility to cancer were found. (32 refs.)

- 77-3489 **Bloom's Syndrome. IV. Sister-Chromatid Exchanges in Lymphocytes.** (Eng.) German, J. (New York Blood Center, 310 East 67th St., New York, NY 10021) Schonberg, S.; Louie, E.; Chaganti, R. S. *Am J Hum Genet* 29(3): 249-255; 1977.

Sister-chromatid exchanges (SCE) in the lymphocytes from 21 individuals with Bloom's syndrome were studied. Healthy men and women were used as controls. The lymphocytes from the controls showed a mean of 9.3 SCE's/cell. Blood samples from the 21 individuals with Bloom's syndrome contained a large proportion of cells that exhibited a clear-cut increase in the number of SCE's (mean of 94.4 SCE's/cell). In addition, some of the dividing lymphocytes from the following five Bloom's syndrome homozygotes (*bl/bl*) were found to exhibit only a few exchanges (mean of 12.3/cell): 11(IaTh), 12(DeTh), 62(JeDix), 64(CrFe), and 71(HaEn). The lymphocytes from these five *bl/bl* individuals were composed of two populations with respect to the number of SCE's per cell, one with an abnormally large amount characteristic of cells from Bloom's syndrome and the other with the small amount characteristic of normal cells. The coexistence of

these two cell populations results in a phenotypic dimorphism, in apparent contradiction to the autosomal recessive mode of inheritance of the syndrome. The results suggest that the defect in Bloom's syndrome may be regulatory in nature giving rise to the observed dimorphism. (13 refs.)

- 77-3490 **Familial Occurrence of Colon and Uterine Carcinoma and of Lymphoproliferative Malignancies. Clinical Description.** (Eng.) Law, I. P. (Hematology, Oncology Section, Dept. Medicine, Womack Army Hosp Fort Bragg, NC 28307) Herberman, R. B.; Oldham, R. K.; Bouzoukis, J.; Hanson, S. M.; Rhode, M. C. *Cancer* 39(3): 1224-1228; 1977.

Carcinoma of the colon occurred in 2 of 3 siblings in one generation, 8 of 19 members of the next generation, and of 41 members of the fourth generation. Uterine cancer or lymphoproliferative malignancies were also found in two family members. It is hypothesized that an autosomal dominant trait involving both sexes is the genetic mechanism responsible. (17 refs.)

- 77-3491 **A Single Gene Does Not Control SV-40 T Antigen Expression in Cancer Families (Meeting Abstract).** (Eng.) Blattner, W. A. (NIH, Bethesda, MD 20014) Blattner, W. A.; Lubiniecki, A. S.; Mulvihill, J. J.; Fraumeni, J. F. *Proc Am Assoc Cancer Res* 18: 63; 1977. (no refs.)

- 77-3492 **Clinical and Cytogenic Aspects of Chromosome Breakage (Meeting Abstract).** (Eng.) Adams, N. J. (No affiliation given) Anderton, L. G. *J Elisha Mitchell Sci Soc* 92(2): 62-63; 1977. (no refs.)

- 77-3493 **Homogenously Staining Regions and Double Minute Chromosomes in Human Neuroblastoma Cell Lines (Meeting Abstract).** (Eng.) Malenbaum, G. B. (Dept. Human Genetics, Sch. Medicine, Univ. Pennsylvania, Philadelphia, PA 19174) Gilbert, F. *Proc Am Assoc Cancer Res* 18: 140; 1977. (1 ref.)

- 77-3494 **Chromosome Replication Patterns in Heteroploid Rat Sarcoma Cells in Primary Culture (Meeting Abstract).** (Eng.) Sklarew, R. J. (New York Univ. Res. Service, Goldwater Memorial Hosp., Roosevelt Island, New York, NY 10044) Hoffman, J.; Post, J. *In Vitro* 13(3): 200; 1977. (no refs.)

- 77-3495 **Effect of Cell Aggregation on Intravenous Tumor Transplantation.** (Eng.) Ryd, W. (Inst. Pathology I, Sahlgrenska sjukhuset, 413 45 Goteborg, Sweden) Hagmar, B. *Acta Pathol Microbiol Scand (A)* 85(3): 405-412; 1977.

The effect of cell aggregates on the pattern of iv induced metastasis of two syngeneic murine tumors was studied. The tumors were the MCG101-AA ascites tumor of C57BL mice and the solid sarcoma MCG1-SS of CBA mice. Aggregates were produced mechanically by centrifugation or chemically by a lectin (wheat germ agglutinin). Compared with well-dissociated suspensions, aggregated suspensions tended to give a greater total metastasis volume in the lungs of recipient mice. Disaggregated suspensions, on the other hand, gave rise to more extrapulmonary metastases. Presumably, aggregates are preferentially retained in lung vessels, but single cells pass through to other sites. The results also show that tumor aggregates are not necessary to induce metastases, nor are aggregates superior to single cells in producing tumor growth when the total metastases yield is considered. (20 refs.)

- 77-3496 **Chromosomes of Three Suspension Lymphoblastoid Cell Lines from Hematosarcoma *Papio hamadryas*.** (Rus.) Markaryan, D. S. (Lab. Experimental Oncology, Inst. Experimental Pathology and Therapy, Acad. Medical Sciences USSR, Suchumi, USSR) Gvaramiya, I. A.; Agrba, V. Z.; Sanguliya, I. A. *Tsitologiia* 19(2): 215-223; 1977.

Three suspension cell lines from the bone marrow (BMPH-1) and spleens (SPH-2 and SPH-3) of hamadryas baboons with hematosarcoma were characterized by the presence of analogous lymphoid-type cells of a high proliferative activity. The modal class of cells in all the three lines was represented by diploids and pseudodiploids, although there was a significant admixture of heterodiploid and subtriploid cells and cells having various types of marker chromosomes. The reconstructed chromosome 1 and satellite chromosomes dominated among the marker chromosomes, indicating their relatively greater mutability. No change was observed in Giemsa banding during a 1-yr cultivation of all three cell lines. The total number of weakly and poorly stained chromosomes in aneuploids was more variable than the number of strongly stained chromosomes. This was presumably a result of the lesser biological significance of the former. (13 refs.)

- 77-3497 **Ultrastructural Features of Fibrosarcomas.** (Rus.) Galil-Ogly, G. A. (Moscow Scientific Res. Inst. Roentgenology and Radiology, Ministry Public Health Russian SSR, Moscow, USSR) Krylov, L. M.; Poroshin, K. K. *Arkhh Patol* 39(3): 51-56; 1977.

Tumor specimens from two patients with soft tissue fibrosarcoma were studied electron microscopically. Both tumors

were poorly differentiated. Morphologically, most of the tumor cells resembled fibroblasts but, unlike typical fibroblasts, they had elements of nonstriated myogenic differentiation. The tumor cells also had a well-developed granular endoplasmic reticulum that was filled with collagen fibrils. (12 refs.)

- 77-3498 **Mesectodermal Leiomyoma of the Ciliary Body: A Tumor of Presumed Neural Crest Origin.** (Eng.) Jakobiec, F. A. (Dept. Ophthalmic Pathology, Armed Forces Inst. Pathology, Washington, DC 20306) Font, R. L.; Tso, M. O.; Zimmerman, L. E. *Cancer* 39(5): 2102-2113; 1977.

Case reports are presented for two patients with benign tumors of the ciliary body. The tumors were diagnosed by light microscopy as being neurogenic tumors, but microscopy showed that they were composed of smooth muscle cells with unusual morphological features. The light microscopic finding of a background fibrillary matrix, which imparted a neural appearance, resulted from the interweaving of myriad cell processes filled with thin cytoplasmic filaments possessing fusiform densities. The perikaryon of the tumor cells was relatively free of filaments and displayed mitochondria and stacks of rough endoplasmic reticulum. It is suggested that these tumors, which combine myogenic and neurogenic characteristics, may constitute a new nosologic entity of myogenic neoplasia. Their occurrence in the ciliary body may be tentatively ascribed to the distinctive embryological origin of the ciliary muscle from the neural crest (mesectoderm). (27 refs.)

- 77-3499 **Abnormalities of Centriole and Cilium Formation in Human Neoplasms Arising in Endocrine Glands (Meeting Abstract).** (Eng.) Horvath, E. (St. Michael's Hosp., Univ. Toronto, Ontario, Canada) Kovacs, K. *Am J Pathol* 86(2): 66a; 1977. (1 ref.)

\* (Rev): 77-3009, 77-3028, 77-3029, 77-3038, 77-3039, 77-3040, 77-3041, 77-3042, 77-3043, 77-3044, 77-3045, 77-3046, 77-3047, 77-3048, 77-3049, 77-3062.

\* (Chem): 77-3087, 77-3089, 77-3097, 77-3098, 77-3099, 77-3105, 77-3106, 77-3107, 77-3110, 77-3118, 77-3119, 77-3149, 77-3154, 77-3159, 77-3175, 77-3177, 77-3205, 77-3206, 77-3207, 77-3208, 77-3210.

\* (Phys): 77-3213, 77-3214, 77-3215, 77-3216, 77-3217, 77-3225, 77-3227, 77-3231.

\* (Viral): 77-3241, 77-3247, 77-3261, 77-3276, 77-3285, 77-3295, 77-3298, 77-3300.

\* (Immun): 77-3324, 77-3329, 77-3330, 77-3342, 77-3343, 77-3344, 77-3345.

\* (Epid): 77-3502, 77-3509, 77-3514, 77-3516, 77-3519, 77-3521, 77-3524, 77-3532.



## EPIDEMIOLOGY AND BIOMETRY

- 77-3500 **Stomach Carcinoma Among Hawaiians and Caucasians in Hawaii.** (Eng.) Wronkowski, Z. (Inst. Oncology, 00-973, Warsaw, Wawelska 15, Poland) Stemmermann, G.; Rellahan, W. *Cancer* 39(5): 2310-2316; 1977.

Pathological studies and survival experience of stomach cancer patients from a high-risk population (native Hawaiians) and from a low-risk population (Caucasians resident in Hawaii) are reported. In addition, comparison is made with data from earlier studies among Hawaiian Japanese, a high-risk population. There are very different survival expectations among the various races in Hawaii: the relative 5-yr survival rate of Hawaiian and Caucasian patients is 6.8%, in contrast to 20.6% among Japanese > 65 yr old and 25% among Japanese < 65 yr. There are also pronounced interracial differences in the ratio of intestinal to diffuse carcinomas in patients: the ratios are 0.76, 1.20, and 1.12 in Japanese, Hawaiians, and Caucasians, respectively. Caucasian men of Portuguese origin suffered a higher incidence of gastric carcinoma (33.6/100,000) than other Caucasians (13.1/100,000). However, this may be consistent with there being an inverse correlation between gastric cancer incidence and socioeconomic prosperity. Overall, the results suggest the existence of a strong host influence upon tumor morphology and prognosis. (18 refs.)

- 77-3501 **Metabolic Epidemiology of Colon Cancer: Fecal Bile Acids and Neutral Sterols in Colon Cancer Patients and Patients with Adenomatous Polyps.** (Eng.) Reddy, B. S. (American Health Foundation, Valhalla, NY 10595) Wynder, E. L. *Cancer* 39(6): 2533-2539; 1977.

Because of the potential significance of bile acids and cholesterol metabolites in the pathogenesis of colon cancer, fecal neutral sterols and bile acids were determined in 31 patients with colon cancer, 13 with adenomatous polyps, 9 with other digestive diseases (spastic colon, irritable colon, diverticulosis, and Crohn's colitis), and 16 normal American or Japanese controls. The fecal excretion of cholesterol, coprostanol, total bile acids, deoxycholic acid, and lithocholic acid was higher in patients with colon cancer or adenomatous polyps than in controls or patients with other digestive diseases. The latter excreted levels of fecal bile acids and cholesterol metabolites comparable to those of the American controls; Japanese controls excreted reduced levels compared with the American controls. These findings suggest that possible interactions between bile acids and cholesterol metabolites and colonic epithelial cells may be relevant in colon carcinogenesis. (26 refs.)

- 77-3502 **The Epidemiology of Colorectal Polyps: Prevalence in New Orleans and International Comparisons.** (Eng.) Correa, P. (Dept. Pathology, Louisiana State Univ. Medical Center, 1542 Tulane Ave., New Orleans, LA 70112) Strong, J. P.; Reif, A.; Johnson, W. D. *Cancer* 39(5): 2258-2264; 1977.

A systematic search for polyps of the large intestine was conducted in autopsy material from New Orleans between 1970 and 1975, in order to establish whether a correlation exists between the prevalence of polyps in a population and incidence of colonic cancer. The prevalence of both hyperplastic and adenomatous polyps is reported, and data from other studies are compared. A close correlation was found between the occurrence of adenomatous polyps and incidence of colonic cancer. The prevalence of these polyps in men of the following groups was: 63% in Hawaiian Japanese, who suffer a very high incidence of colonic cancer; 37% in New Orleans black and 36% in New Orleans whites, who both have high incidences of colonic cancer; 14% in Japanese from Miyagi, who suffer a low incidence of colonic cancer; and 11% in Columbians, who also suffer a low incidence of colonic cancer. The respective values in hyperplastic polyps was 73%, 15%, 14%, 3%, and 11%. The type and distribution of the polyps found are described further, and the significance of the established correlation between adenomatous polyps and colonic neoplasms is discussed. It is concluded that adenomatous polyp and colon adenocarcinoma are causally associated. (26 refs.)

- 77-3503 **Bowel Transit-time and Stool Weight in Populations with Different Colon-cancer Risks.** (Eng.) Glober, G. A. (Japan-Hawaii Cancer Study, Kuakini Medical Center, 347 N. Kuakini St., Honolulu, HA 96817) Kamiyama, S.; Nomura, A.; Shimada, A.; Abba, B. C. *Lancet* 2(8029): 110-111; 1977.

Bowel transit time and stool wt were compared in the Japanese residents of Hawaii and natives of Japan. The bowel transit times were similar in both groups; stools from the Hawaiian Japanese weighed significantly less than the specimens from Japan. Identification of the causes of stool-weight differences between these two ethnically similar groups may clarify the possible relationship between stool wt and cancer risk. (8 refs.)

- 77-3504 **Role of Nutrition in the Etiology of Breast Cancer.** (Eng.) Miller, A. B. (NCIC Epidemiology

Unit., Univ. Toronto, 121 St. Joseph St., Toronto, M5S 2R9, Ontario, Canada) *Cancer* 39(6): 2704-2708; 1977.

International differences in breast cancer morbidity and mortality and studies of migrating populations point to the overriding importance of environmental factors in the etiology of breast cancer. Factors directly or indirectly associated with ovarian activity do not appear to explain international differences. Population correlation studies have indicated that much of the difference is explicable on the basis of nutritional factors, particularly high total fat intake. Animal experimental studies confirm the importance of a high fat diet, possibly mediated through prolactin. Other indirect support for the nutritional hypothesis is supplied by changing incidence rates in Iceland, possibly correlated with changing nutritional practices and the association of breast cancer with wt and possibly also with height. The association with height (which would suggest nutritional effects mediated through childhood diet) has not been supported by a Canadian study. Direct investigation of the association between nutrition and breast cancer is difficult because of problems in dietary methodology. Nevertheless, the results of the Canadian case-control study provide support for the importance of high fat intake. Further studies are undoubtedly required, however, before specific recommendations for dietary modification can be made. (20 refs.)

77-3505    **Nutrition and the Etiology and Prevention of Breast Cancer.** (Eng.) Wynder, E. L. (American Health Foundation, Naylor Dana Inst. Disease Prevention, Valhalla, NY 10595) MacCornack, F.; Hill, P.; Cohen, L. A.; Chan, P. C.; Weisburger, J. H. *Cancer Detect Prevent* 1(2): 293-310; 1976.

Evidence from epidemiological and animal studies that relates the consumption of dietary fat to an increased risk of breast cancer, particularly its postmenopausal form, is reviewed. A high-fat diet uniformly increases the incidence of both spontaneous and chemically induced breast cancer in animal model systems. US white women consume about 130-150 g/day of dietary fat, compared to an intake of about 40 g/day for Japanese women. The incidence of breast cancer in these two groups is 22/100,000 and 4/100,000, respectively. However, migrant Japanese, especially those of the second generation, demonstrate an increased incidence. High dietary fat may influence endocrine balance, as elevated prolactin levels are associated with high breast cancer incidence in animal models and in humans. Puberty may be a particularly susceptible period with regard to breast cancer induction by dietary fat. (74 refs.)

77-3506    **Is Gastric Cancer Related to Diet? (Meeting Abstract).** (Eng.) Marquardt, H. (Naylor Dana

Inst., American Health Foundation, Valhalla, NY 10595) Weisburger, J. H. *Gastroenterology* 72(5/Part 2): 1098; 1977. (no refs.)

77-3507    **Epidemiologic Characteristics of Benign Breast Disease.** (Eng.) Nomura, A. (Training Center for Public Health Res., Box 2067, Hagerstown, MD 21740) Comstock, G. W.; Tonascia, J. A. *Am J Epidemiol* 105(6): 505-512; 1977.

A retrospective study of benign breast disease in a general population was conducted to determine if the risk factors associated with fibroadenoma and cystic breast disease were similar to those reported for breast cancer. The study population consisted of 320 white women (20-49 yr old) who had had benign breast disease (275 cystic disease and 45 fibroadenoma) and 320 age-matched controls. More cystic disease patients than controls had the following breast cancer-associated characteristics: higher socioeconomic status, fewer pregnancies, and a lack of association with lactation patterns. Nulliparity, late natural menopause, and a maternal history of breast cancer were also more common among cystic cases than controls, although these differences could have occurred by chance. Cystic disease cases and controls did not differ with respect to other factors associated with breast cancer, such as early age at menarche, late age at first pregnancy, and negative history of artificial menopause resulting from surgery or radiation therapy. Fibroadenoma was not associated with most of the risk factors of breast cancer. Evidence from this and previous studies indicates more epidemiologic similarities than dissimilarities between cystic breast disease and breast cancer. The findings also suggest that fibroadenoma and breast cancer may be entirely separate entities. (28 refs.)

77-3508    **Unilateral Breast-Feeding and Breast Cancer.** (Eng.) Ing, R. (G. W. Hooper Foundation, 1699 HSW, Univ. California, San Francisco, CA 94143) Ho, J. H.; Petrakis, N. L. *Lancet* 2(8029): 124-127; 1977.

The hypothesis that the unsuckled breast may have an altered risk of cancer development was investigated using the records of all breast-cancer patients treated in radiotherapy departments of Hong Kong hospitals between 1958 and 1975. In addition, a search was made for breast cancer patients from the Tanka boat people (who by custom breast-feed with the right breast only) and for any patient with a history of breast-feeding on one side. The overall left/right ratio for 2,372 patients with unilateral breast cancer was 0.97, an equal distribution. Of 73 patients who had breast-fed unilaterally, 27/34  $\geq$  55 yr old and 19/39 < 55 yr old had a cancer in the unsuckled breast. Comparisons of patients who had nursed



unilaterally with nulliparous patients and with patients who had borne children but had not breast-fed also indicated a highly significant increased risk of cancer in the unsuckled breast. According to this study, in postmenopausal women who have breast-fed unilaterally, the risk of cancer is three to four times higher in the unsuckled breast. (22 refs.)

- 77-3509 **Chronic Mastopathy and Breast Cancer: A Follow-up Study.** (Eng.) Kodlin, D. (Dept. Biometry, LSU Medical Center, 1542 Tulane Ave., New Orleans, LA 70130) Winger, E. E.; Morgenstern, N. L.; Chen, U. *Cancer* 39(6): 2603-2607; 1977.

A total of 2,900 cases of benign breast lesions diagnosed by biopsy between 1948 and 1973 at the Kaiser Foundation Hospital, Oakland, California, were followed for cancer development for an av of 7 yr. When classified according to traditional diagnostic criteria, the annual cancer incidence rate per 1,000 (person-year rate) ranged from 2.7 to 7.9 and appeared elevated in comparison with expectations based on the Third National Cancer Survey, San Francisco Bay Area. Increased relative risks were noted for fibroadenoma (7.0), adenosis or fibrosing adenosis (5.0), and intraductal papilloma (5.0). Of the benign biopsies, 2,411 were scored for atypia by the Black-Chabon method. There was an upward trend in breast cancer incidence as the atypia score rose, a finding that confirms conclusions from a 1972 retrospective case-control study. (13 refs.)

- 77-3510 **The Increasing Incidence of Breast Cancer in Alberta 1953-1973.** (Eng.) Grace, M. (Provincial Cancer Hosps. Board, 11560 University Ave., Edmonton, Alberta, Canada T6G 1Z2) Gaudette, L. A.; Burns, P. E. *Cancer* 40(1): 358-363; 1977.

The incidence of female breast cancer in Alberta increased steadily by 1 case/100,000/yr from 1953 to 1973 to a current rate of 68.6/yr when adjusted to the 1950 US population. Incidence rates of breast cancer in Alberta and Saskatchewan were identical after population adjustment. The incidence rose in women > 40, implicating an increase in postmenopausal type breast cancer. Birth cohort analysis showed increased age-specific incidence rates in middle-aged women occurring in successive cohorts from 1903 to 1918, a result similar to that found in Saskatchewan, Connecticut, and Finland. Possible etiological factors involved in these incidence changes are discussed; a detailed analysis of specific etiological factors is currently underway on > 3,000 patients with malignant or benign breast disease who were examined from 1971 to 1974. (25 refs.)

- 77-3511 **Changing Trends in Morbidity and Mortality in Endometrial Carcinoma.** (Eng.) Christopherson, W. M. In: *Endometrial Carcinoma and Its Treatment: The Role of Irradiation, Extent of Surgery, and Approach to Chemotherapy.* Gray, L. A., ed. (Springfield, IL: C. C. Thomas) pp. 9-18; 1977.

Discussion is made of the following aspects of endometrial carcinoma: incidence, risk factors, the effects of race on incidence, the effects of exogenous estrogens, and mortality experience. There was a small (15.9%: not significant) increase in the incidence of the disease in Louisville, Kentucky, between 1953 and 1967. This increase was seen only among the white population; a slight decrease in incidence was seen among the black population. Data from a small number of patients in the Louisville registry indicated a survival of 70.9% at 5 yr and 55.8% at 10 yr. (14 refs.)

- 77-3512 **Liver Tumors and Contraceptive Steroids: Experience with the First One Hundred Registry Patients.** (Eng.) Christopherson, W. M. (Dept. Pathology Univ. Louisville Sch. Medicine Health Sciences Center, Louisville, KY 40201) Mays, E. T. *J Natl Cancer Inst* 58(2): 167-171; 1977.

There is evidence that steroid contraceptives are involved in the sudden increase of benign liver tumors in young women; the evidence is less compelling in the case of hepatomas. It is suggested that the estrogen component of the steroid regimens, rather than the progestin component, is the cause of tumor induction. Clinical findings for the first 100 patients accessioned into the registry of liver tumors at the University of Louisville since 1973 are summarized. The average was 29.5 yr for 40 patients with adenoma, 29.8 yr for 44 patients with focal nodular hyperplasia (FNH), and 30.4 yr for 13 patients with hepatoma. Of the 40 patients with adenoma, 15 had taken mestranol (M). Of the 44 FNH patients, 23 had taken M, 1 had taken EE, 5 had taken M + EE, and 2 had taken premarin. Of the 13 hepatoma patients, 4 had taken M, 3 had taken EE, 1 had taken M + EE, and 1 had taken premarin. (19 refs.)

- 77-3513 **Estrogens, Progestogens and Endometrial Cancer.** (Eng.) Gambrell, R. D. (Dept. Obstetrics and Gynecology, Wilford Hall USAF Medical Center, Lackland AFB, TX) *Reprod Med* 18(6): 301-306; 1977.

The incidence of endometrial cancer and the history of steroid therapy are described for postmenopausal patients at the Wilford Hall USAF Medical Center, Texas. Adenocarcinoma of the endometrium was diagnosed in 7/2,300 post-

menopausal patients; 6/7 patients had received estrogen therapy. Of these six, estrogen had been the only therapy in five, yielding an incidence of endometrial cancer of 4.7/1,000 in this group. The other endometrial malignancy occurred in 1/1,240 patients who had received both progestogen and estrogen; 1/510 untreated postmenopausal women also developed adenocarcinoma of the endometrium. It is tentatively concluded that there is some increased risk of endometrial cancer from estrogen therapy for menopause, but that this risk may not be as great with cyclic estrogens. (18 refs.)

- 77-3514 **The Disease in Young Women.** (Eng.) Silverberg, S. G. In: *Endometrial Carcinoma and Its Treatment: The Role of Irradiation, Extent of Surgery, and Approach to Chemotherapy*. Gray, L. A., ed. (Springfield, IL: C. C. Thomas) pp. 19-27; 1977.

The incidence and treatment of endometrial carcinoma in younger women are reviewed. Premenopausal women currently account for 10%-25% of endometrial cancer victims. Although in one series only 5% of endometrial carcinoma patients with pure adenocarcinoma were premenopausal, 35%-40% of those with carcinomas in which squamous elements were present (adenocanthoma and mixed adenosquamous carcinoma) were premenopausal. A typical clinical profile of low fertility, obesity, hypertension, and diabetes, frequently described in older patients with carcinoma of the endometrium, is also found in patients < 40 yr of age. (14 refs.)

- 77-3515 **Contacts Between Young Patients with Hodgkin's Disease. A Case-control Study.** (Eng.) Smith, P. G. (D.H.S.S. Cancer Epidemiology and Clinical Trials Unit, Dept. Regius Professor Medicine, Univ. Oxford, 9 Keble Road, Oxford, England) Kinlen, L. J.; Pike, M. C.; Jones, A.; Harris, R. *Lancet* 2(8028): 59-62; 1977.

A study of contacts among 87 Hodgkin's disease patients in a defined residential area failed to reveal any evidence that the disease was passed from one to another. Only one significant association indicated that a patient was infective 10 to 5 yr before diagnosis and from diagnosis to 2 yr afterwards, but this association may have arisen by chance. (13 refs.)

- 77-3516 **Hodgkin's Lymphoma in Childhood (Histopathological Analysis of 157 Hodgkin's Cases and Its Occurrence Among the Childhood Malignant Tumors).** (Eng.) Tel, N. (Hacettepe Univ., Children's Medical Center, Dept. Pediatric Pathology, Turkey) Tinaztepe, B.; Tinaztepe, K. *Kanser* 6(2): 132-146; 1976.

The histopathologic records of 157 Turkish children (aged 1-16 yr) with Hodgkin's disease diagnosed within the last 14 yr were reviewed. Nine of the children underwent staging laparotomy. Malignant lymphomas are the most common neoplasm (22.2% incidence) and Hodgkin's disease is the most common lymphoma (47%) among Turkish children. The cervical lymph nodes were involved in 75% of the patients. The male/female ratio was 3.3:1, and the peak incidence was in the age group 6-8 yr. The most frequent subtype, both in the original biopsy and after the staging laparotomy, was mixed cellularity (59.6%), followed by lymphocyte depletion (26%), lymphocytic predominance (22%), and nodular sclerosis (17%). In general, the subtypes did not change upon subsequent biopsies or at laparotomy performed up to 5 mo later. No significant sex difference was noted among the subtypes. Associated glomerulopathy and amyloides were found occasionally. These findings are compared with data from other countries. (42 refs.)

- 77-3517 **A Statistical Analysis of 704 Childhood Tumors of the Nervous System (Meeting Abstract).** (Eng.) Becker, L. E. (Hosp. for Sick Children, Toronto, Ontario, Canada) Yates, A. J. *Am J Pathol* 86(2): 57a; 1977. (no refs.)

- 77-3518 **Occurrence of Malignant Tumors in Elderly Patients.** (Ger.) Noltenius, H. (Allgemeines Krankenhaus St. Georg, Lohmühlenstrasse 5, D-2000 Hamburg 1, W. Germany) Giersch, H.; Haake, A.; Raydt, H. J.; Buchholz, M. *Med Klin* 72(10): 391-398; 1977.

Macroscopic findings in 2,385 autopsies were analyzed for the frequency and distribution of malignant tumors. The frequency of cancer of the stomach, colon, prostate, and breast increased with increasing age, but that of cancer of the lung and uterus decreased with age. Severe tumor-independent disease was an almost constant finding. The overall frequency of malignant tumors decreased with increasing age. (14 refs.)

- 77-3519 **Benign Tumors of the Lungs.** (Rus.) Aitakov, Z. N. (Section Oncology, Khabarovsk Medical Inst., Khabarovsk, USSR) Efremenko, V. M.; Monin, M. I.; Gibradze, O. T. *Grudn Khir* (2): 99-101; 1977.

From 1959 to 1975, 66 patients with benign tumors of the lungs underwent treatment in Khabarovsk, USSR. The group comprised 31 men and 35 women aged 18-64 yr. The tumor was located in the right lung in 43 patients and in the left lung in 23. Twenty-five patients had epithelial tumors (24 adenomas and 1 papilloma) and 39 patients had nonepithelial



tumors (32 hamartomas and chondromas, 1 lipoma, 2 fibromas, 2 lymphomas and plasmocytomas, and 2 neurilemmomas). Roentgenological examination showed bronchostenosis with hypoventilation in 7 patients, atelectasis in 11 patients, and pleuritis in 4. Bronchoscopy produced a correct diagnosis in 17/21 patients with endobronchial tumor growth. Treatment of choice was surgery. Three patients refused to undergo operation. Of 63 patients, 7 underwent pneumonectomy, 3 bilateral lobectomy, 12 lobectomy, 5 bi- and segmentectomy, 9 clinoid resection, 5 bronchotomy, and 22 tumor enucleation. Two patients died immediately after surgery (1 of bronchial stump failure and 1 of acute fibrinolysis). Forty-seven patients were followed for 1-5 yr: 6 died (1 of metastases of lung cancer), but the rest remained in good health. (3 refs.)

**77-3520 Incidence, Diagnosis, and Surgical Treatment of Pulmonary Carcinoma in Leningrad (1968-1975).** (Rus.) Drukin, E. Ia. (Leningrad Municipal Oncological Dispensary, Leningrad, USSR) Iaritsyn, S. S.; Golikova, M. A. *Klin Med* 55(3): 120-124; 1977.

The occurrence and treatment of lung cancer in Leningrad are reviewed. From 1968 to 1975, lung cancer was detected in 11,620 individuals (9,000 men, 2,521 women). The male:female ratio was approx 1:1 in patients < 29 yr, 6:1 in the 40- to 59-yr-old age group, and 2:1 in patients > 70 yr. Radical surgery was performed in 1,818 patients, 228 patients underwent exploratory thoracotomy, 1,629 patients refused to undergo surgery, and 7,945 patients with far-advanced tumors had contraindications to surgery. (3 refs.)

**77-3521 Malignancies Associated with Renal Transplantation.** (Eng.) Penn, I. (1055 Claremont St., Denver, CO 80220) *Urology (Suppl)* 10(1): 57-63; 1977.

An increased incidence of cancer occurs in renal homograft recipients. Malignancies may be inadvertently transplanted with the kidney from donors with cancer, or they may arise de novo at some time after transplantation. The latter tumors occur an av of 34 mo after the operation. The most common tumors are carcinomas of the skin and lip, lymphomas (mostly reticulum cell sarcomas), and carcinomas of the uterine cervix. The lymphomas have a marked predilection for the CNS. Besides conventional cancer therapy, reduction or cessation of immunosuppression may be warranted. The development of malignancies is not a contraindication to renal transplantation, since the overall death rate from cancer in kidney homograft recipients is low. (28 refs.)

**77-3522 Splenectomy and Subsequent Mortality in Veterans of the 1939-45 War.** (Eng.) Robinette, C.

D. (Medical Follow-up Agency, Natl. Acad. Sciences-Natl. Res. Council, 2101 Constitution Ave., N.W., Washington D.C., 20419) Fraumeni, J. F. *Lancet* 2(8029): 127-129; 1977.

A follow-up of 740 American servicemen who underwent splenectomy because of trauma between 1939 and 1940 revealed no increased risk of cancer, although an excess mortality from pneumonia and eschismic heart disease was found. It is suggested that this was because cell-mediated immunity was not impaired in the asplenic group, as it is in other immunodeficient patients who are predisposed to cancer. (12 refs.)

**77-3523 Increase in the Number of Cancer Deaths in the United States.** (Eng.) Devesa, S. S. (Field Studies and Statistics, NCI, Landow Building Room B506, 7910 Woodmont Ave., Bethesda, MD 20014) Schneiderman, M. A. *Am J Epidemiol* 106(1): 1-5; 1977.

Using 1930 as a base line, the increase in age-specific cancer mortality during the period 1930 to 1970 has been 26.8%, as opposed to the 180.5% increase that is obtained without considering the changes in age distribution (ie, a greater proportion of older people) and the population increase. Subgroups of the population should now be examined for changing incidences that may reflect environmental carcinogenesis. (7 refs.)

**77-3524 On the Incidence of Malignant Tumors in Routine Autopsies.** (Eng.) Niedobitek, C. (Pathologisches Institut am Stadt. Auguste-Viktoria-Krankenhaus, Rubensstrasse 125, D-1000 Berlin 41, W. Germany) Niedobitek, F.; Pfitzinger, H.; Voss, J. D.; Griesser, G. *Z Krebsforsch* 88(2): 157-184; 1977.

A detailed report is presented of a survey of 4,911 malignant tumors found in 15,384 routine autopsies performed in Lubbeck, West Germany, over the period 1960-1971. The frequency, location, age and sex distributions, and histological classification of the tumors were analyzed. Most of the malignant tumors were located in the digestive system (26.2%) followed by the urogenital system (23.4%) and the respiratory system (18.7%). The highest tumor rates for individual organs in men were bronchi and lungs (28.3%) and stomach (13.7%); in women the highest organ rates were for the uterus (15.2%), mammary glands (12.6%), and stomach (10.2%). (39 refs.)

**77-3525 Cancer in Yogyakarta, Indonesia: Relative Frequencies.** (Eng.) Soeripto (Dept. Pathology,

Gadjah Mada Univ., Yogyakarta, Indonesia) Jensen, O. M.; Muir, C. S. *Br J Cancer* 36(1): 141-148; 1977.

Some 1,220 male and 2,102 female cases of malignant neoplasms diagnosed histologically at the Department of Pathology of the Gadjah Mada University in Yogyakarta, Indonesia, during the period 1970-1973 were analyzed. The most frequent tumors among men were nasopharyngeal cancer (21.8%) and skin cancer (17.6%). Among women, genital cancers were the most frequent: 25.7% of all tumors were located in the cervix uteri, but there was a high proportion of chorioepithelioma (3.7%), ovarian cancer (7.9%), and other uterine cancer (4.4%). Other frequent sites were the breast (17.0%), skin (9.6%), and nasopharynx (7.9%). (16 refs.)

**77-3526 Low Cancer Incidence and Mortality in Utah.** (Eng.) Lyon, J. L. (Dept. Family and Community Medicine, Div. Epidemiology, Univ. Utah Coll. Medicine, 50 N. Medical Drive, Salt Lake City, UT 84132) Gardner, J. W.; Klauber, M. R.; Smart, C. R. *Cancer* 39(6): 2608-2618; 1977.

Utah cancer mortality for the period 1950-1969 and morbidity for the period 1966-1970 are reported. Utah had 18% fewer cases of cancer than expected based on the Third National Cancer Survey and 24% fewer cancer deaths than expected based on national mortality data. Cancer sites associated with cigarette smoking and alcohol use accounted for nearly half of these differences. Several major sites not strongly associated with smoking showed a lower incidence and mortality than expected. These included the pancreas, colon, rectum, female breast, uterine cervix, and ovary. A marked excess occurrence above expectations was observed for cancer of the lip (6.6 cases/100,000 population). Some possible explanations of these findings are discussed, including the unique religious and cultural background of Utah residents. (47 refs.)

**77-3527 Some Observations Concerning the Demographic and Geographic Incidence of Carcinoma of the Lip and Buccal Cavity.** (Eng.) Szpak, C. A. (Evelyn L. Overton Hematology-Oncology Res. Lab., Dept. Internal Medicine, Southwestern Medical Sch., 5323 Harry Hines Blvd., Dallas, TX 75235) Stone, M. J.; Frenkel, E. P. *Cancer* 40(1): 343-348; 1977.

The geographic and demographic data obtained during the Third National Cancer Survey provided a perspective on etiologic factors and incidence trends for cancers of low frequency. The incidence of cancer of the lip, oral cavity, and skin from this survey was compared with similar studies in 1947, and intraregional patterns in one area of the Third National Cancer Survey (Dallas-Fort Worth) were evaluated. The age-adjusted annual incidence of lip cancer in white

men in this area was 11.5/100,000 (based on the 1950 population standard), twofold greater than that in the geographic area (Iowa) with the second highest incidence and approximately threefold greater than that in all the other areas. The incidence in white women was only 8% that in white men. Intraregional differences were also seen, with the incidence of lip cancer in Fort Worth men being 50% greater than in a similar population in Dallas. Incidence trends over the past two decades reveal a significant decline in the incidence of oral cavity cancer and a slight decrease in lip cancer. Comparisons of the incidence of lip cancer did not correlate with skin cancer incidence or with geographic latitude in the other survey areas. The studies fail to support the classical implication of actinic radiation as the primary etiologic factor in lip cancer incidence. (16 refs.)

**77-3528 The Etiology of Bladder Cancer from the Epidemiological Viewpoint.** (Eng.) Miller, A. B. (Epidemiology Unit, Natl. Cancer Inst. Canada, Univ. Toronto, 121 St. Joseph St., Toronto, Ontario, M5S 2R9, Canada) *Cancer Res* 37(8/part 2): 2939-2942; 1977.

A case-control study in two geographically distinct areas of Canada indicated a correlation between cigarette smoking and bladder cancer, as well as an increased risk of bladder cancer in coffee drinkers. However, it is suggested that other substances to which the population is exposed may also contribute to the increased risk of bladder cancer. (12 refs.)

**77-3529 Quantification of the Role of Smoking and Chewing Tobacco in Oral, Pharyngeal, and Esophageal Cancers.** (Eng.) Jayant, K. (Tata Memorial Center, Parel, Bombay 400 012, India) Balakrishnan, V.; Sanghvi, L. D.; Jussawalla, D. J. *Br J Cancer* 35(2): 232-235; 1977.

The proportions of oral, pharyngeal, and esophageal cancers attributable to tobacco smoking and/or chewing and expressed as etiologic fractions were estimated. Based on data for 2,005 cancer patients and an equal number of controls, the overall etiologic fractions were 70% for cancer of the oral cavity, 84% for cancer of the oropharynx, and about 75% for cancer of the hypopharynx and larynx. The fraction was only 50% for esophageal cancer, which indicates that another factor(s) plays an equal role in its etiology. At each of the sites studied, tobacco smoking and chewing acted synergistically, although in varying degrees. (9 refs.)

**77-3530 Cancer of the Oral Cavity, Pharynx/Larynx and Lung in North Thailand: Case-Control**



**Study and Analysis of Cigar Smoke.** (Eng.) Simarak, S. (Faculty Medicine, Chiang Mai Univ., Chiang Mai, Thailand) de Jong, U. W.; Breslow, N.; Dahl, C. J.; Ruckphaopunt, K.; Scheelings, P.; MacLennan, R. *Br J Cancer* 36(1): 130-140; 1977.

The unusually high frequency of laryngeal cancer in men (18% of all histologically diagnosed cancers) and a sex ratio of unity for lung cancer in Northern Thailand were further explored in a hospital-based case-control study in Chiang Mai. A comparison was made of patients with cancers of the oral cavity (including oropharynx), larynx, hypopharynx, and lung with controls in relation to smoking and chewing habits. Statistical analysis indicated that chewing betel is strongly associated with the occurrence of oral cancer in both sexes and with cancer of the laryngeal region in men. No factors were strongly linked to lung cancer in men, but in women, urban residence and miang (fermented wild tea leaves + salt, ginger, fried coconut, or garlic) chewing were associated with lung cancer. Analysis of smoke from the two main types of cigars smoked in the region showed that both had high tar content, but there were marked differences in pH. Smoking cigars with alkaline smoke and high tar increased the risk for laryngeal cancer in men, but smoking cigars with acid smoke and high tar together with manufactured cigarettes increased the risks for lung cancer. These increased risks were not, however, statistically significant. (21 refs.)

**77-3531 Esophageal Cancer in Ille-et-Vilaine, France, in Relation to Alcohol and Tobacco Consumption: Multiplicative Risks.** (Fre.) Tuyns, A. J. (International Agency Res. Cancer, 150, cours Albert-Thomas, F 69008, Lyon Cedex 2, France) Pequignot, G.; Jensen, O. M. *Bull Cancer (Paris)* 64(1): 45-60; 1977.

A retrospective case-control study of 200 men with esophageal cancer and 778 population controls was carried out in Ille-et-Vilaine, France. The logarithms of the relative risks of developing the disease increase linearly with daily consumption of alcohol or tobacco. The combined effects of both fit with a multiplicative model that is proposed. This model could be applicable to other situations. It explains the sex ratio and the urban/rural differences observed in Ille-et-Vilaine. The practical implications for public health purposes are discussed briefly. (22 refs.)

**77-3532 Lung Cancer Type in Relation to Cigarette Dosage.** (Eng.) Weiss, W. (Div. Occupational Medicine, Hahnemann Medical Coll. and Hosp., 230 N. Broad St., Philadelphia, PA 19102) Altan, S.; Rosenzweig, M.; Weiss, W. A. *Cancer* 39(6): 2568-2572; 1977.

In a retrospective study of 1,228 white men with histologically confirmed bronchogenic carcinoma, the frequency of squamous cell carcinoma rose from 39.9% in men aged < 50 yr to 60% in men aged  $\geq 70$  yr. The frequency of undifferentiated carcinoma, adenocarcinoma, and alveolar cell carcinoma decreased with increasing age. Since the distribution of cell types was similar in the 73% of the men aged 50-69 yr, the relationship of cancer type to daily cigarette consumption was studied in this group. The proportion of squamous cell carcinomas increased from 48% in men who smoked < 2 cigarettes/day to 61% in those who smoked  $\geq 40$  cigarettes/day. Comparison with other studies showed conflicting results. (13 refs.)

**77-3533 Unsuspected Exposure to Asbestos and Bronchogenic Carcinoma.** (Eng.) Martischnig, K. M. (Queen Elizabeth Hosp., Gateshead, Tyne and Wear NE6 6SX, England) Newell, D. J.; Barnsley, W. C.; Cowan, W. K.; Feinmann, E. L.; Oliver, E. *Br Med J* 1(6063): 746-749; 1977.

A study was conducted to determine whether unsuspected asbestos exposure in the absence of asbestosis predisposes to carcinoma, whether smoking is relevant in this context, and whether this type of cancer differs in any way from other bronchial carcinomas. Two hundred and fifty men admitted to a thoracic surgical center and matched controls were questioned in detail about their occupations after leaving school and their smoking habits. Of 201 men with bronchial carcinoma, 58 gave a history of occupational asbestos exposure; only 29/201 controls gave such a history. This difference was statistically significant. The usual association of bronchial carcinoma with heavy smoking was observed, but asbestos exposure increased the risk of carcinoma whatever the smoking level. These results are consistent with the hypothesis that asbestos exposure and smoking act independently in causing bronchial carcinoma. The patients with carcinoma who had been exposed to asbestos presented 3 yr earlier than those who had not been exposed. Tissue was obtained from 161 patients for histological examination. Anaplastic carcinoma was nearly twice as common among the exposed men as among the unexposed. However, the histologic types vary depending on whether a series is derived from biopsy, operation, or necropsy. Because regulations have eliminated the risk of exposure to workers in scheduled industries, asbestos-induced diseases will probably be increasingly found among those who have had incidental exposure. It is therefore important to take a full occupational history. (14 refs.)

**77-3534 Epidemiology of Lung Cancer in Italy.** (Eng.) Saracci, R. (Unit Epidemiology and Biostatistics, International Agency Res. Cancer, 150 Cours Albert

Thomas, 69372 Lyon Cedex 2, France) *IARC Sci Publ* (16): 205-215; 1977.

Data related to lung cancer epidemiology in Italy were analyzed for the period 1951-1971. In 1951, lung cancer deaths were 1% of all deaths in men and 0.3% in women, but in 1971, lung cancer contributed to 4.5% of total deaths in men and 0.8% in women. There is a gap in lung cancer mortality rates between the cohort born in 1890 and that born in 1900. As far as men are concerned, this would fit well with the period in which cigarette smoking started, but this explanation does not account for the gap for women nor for the successive increase in female mortality with each successive cohort. The relationship between male regional lung cancer mortality rates in 1970/71 was correlated with six demographic and environmental variables. These variables are, in decreasing order of importance: (1) cigarette consumption per person and (2-6) density of population, sulfur dioxide production, chemical industry, cars, and mineral industry. The data available for current analyses cannot provide an interpretation of what the urban factor (represented by the variable population density) may be, particularly in connection with urban environmental pollution from heating, industrial, and car sources. (8 refs.)

**77-3535 Epidemiology of Lung Cancer in the United States.** (Eng.) Higgins, I. T. (Dept. Epidemiology, Univ. Michigan Sch. Public Health, 109 Observatory St., Ann Arbor, MI 48104) *IARC Sci Publ* (16): 191-203; 1977.

The temporal and spatial distribution of lung cancer in the US is reviewed. Mortality from lung cancer is increasing in all age groups, but the upturn appears earliest in the 35- to 44-yr-olds. Consistently high rates for white men are seen along the Gulf Coast, southeast Atlantic coast, Hudson River, in northern New Jersey, and in New York City. Among nonwhites, high rates are seen in Maryland, Pennsylvania, Ohio, Indiana, Illinois, Iowa, and Wisconsin. There is a higher lung cancer rate among nonwhite than among white men. The main cause of lung cancer is cigarette smoking. There is also an urban factor in lung cancer, but whether carcinogenic pollutants, known to be present in city air, are responsible is much less certain. Recent studies have attempted to relate lung cancer mortality to possible arsenic or benz(a)pyrene exposure, but the results are not conclusive. Evidence of genetic factors in lung cancer has also been presented. Recent studies have indicated that the capacity for aryl hydrocarbon hydroxylase induction may differ in lung cancer patients and other persons. If these findings are substantiated, they may lead to a screening method for identifying a high risk group for respiratory cancer. (23 refs.)

**77-3536 Epidemiology of Lung Cancer in Scandinavia.** (Eng.) Saxen, E. (III Dept. Pathology, Univ.

Helsinki, Haartmaninkatu 3, Helsinki 25, Finland) Teppo, L.; Hakulinen, T. *IARC Sci Publ* (16): 217-228; 1977.

The incidence of lung cancer in Denmark, Finland, Iceland, Norway, and Sweden was analyzed in relation to time trends, cigarette consumption, and urban/rural factors. The incidence is highest in Finland and lowest in Iceland. One-third of all cancer cases in men in Finland originate in the lungs, compared with 16% in Denmark, 10%-11% in Sweden and Norway, and 6% in Iceland. In women, the proportion of lung cancer from all malignant neoplasms is 2.4%-3.4%. The age-specific incidence curves for lung cancer in men are similar for all countries; there is a peak incidence at 70-74 yr in Finland, Denmark, and Sweden and at 65-69 yr in Norway. The urban: rural ratios of the age-adjusted incidence rates are characterized by a low figure for Finnish men and a high figure for Danish women compared to other countries. The correlation between smoking in 1935 and the incidence of lung cancer in 1965 is fairly good. The percentage of heavy smokers in Finland is much greater than that in Norway. It is concluded that the large differences in the incidences of lung cancer among Scandinavian men can be largely accounted for by differences in cigarette consumption and smoking habits in the past. (15 refs.)

**77-3537 Spatial Analysis of Mortality Caused by Malignant Neoplasms of the Breast and Lungs in the Population of Cracow.** (Pol.) Cholewka-Cabaj, K. (Zaklad Epidemiologii Instytutu Medycyny Społecznej AM, 31-511 Krakow, ul. Kopernika 7, Poland) *Nowotwory* 27(1): 73-80; 1977.

Information from the 1974 death certificates was used to analyze the distribution of mortality due to malignant neoplasms within the city of Cracow. The highest mortality was found in the center of the city (4,600 women, 12,600 men), whereas in the surrounding areas it was lower (1,500 women, 2,600 men). The most frequent cause of death was lung cancer in men and breast cancer in women, followed by stomach cancer for both sexes. Breast cancer mortality was highest in the center of the city (4,600) and lowest in the section of Nowa Huta (1,600). However, in Nowa Huta the mortality was higher for premenopausal women than in center-city (8,000/3,000). Mortality from lung cancer was highest in the section of Podgorze and center-city, where there is a high level of air pollution and tobacco consumption and a high percentage of industrial workers. (13 refs.)

**77-3538 Demographic Variation in Cancer in Relation to Industrial and Environmental Influence.** (Eng.) Macdonald, E. J. (Dept. Epidemiology, Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77030) *Environ Health Perspect* 17: 153-166; 1976.



Mortality data (from 183,064 deaths in the period 1940-1969) were analyzed with respect to geographic localities within the city of Houston, Texas. Meteorological and air pollution data were available for each of the 16 selected localities. A significant comparison of mortality data for white (excluding Spanish-surnamed), nonwhite, and Spanish-surnamed populations was possible for certain localities. Evidence was found that exposure to industrial pollutants increased local mortality from cancer of the respiratory tract and from heart disease. Age-adjusted rates for mortality from respiratory malignancies varied from 26/100,000 to 46/100,000 among the total population at different localities. (12 refs.)

- 77-3539 **Factory Populations Exposed to Crocidolite Asbestos--a Continuing Survey.** (Eng.) Jones, J. S.; Polley, F. D.; Smith, P. G. In: *Environmental Pollution and Carcinogenic Risks*. (Lyon: International Agency for Research on Cancer); IARC Scientific Publications No. 13, INSERM Symposia Series Vol. 52, pp. 117-120; 1976.

Data on deaths from mesothelioma among workers in gas mask factories in Nottingham, Preston, and Birmingham are presented. It is indicated that exposure to crocidolite fiber, even in small concentrations, increased the risk of cancer 20-40 yr later. The majority of cases occurred in women, and autopsy studies on lung tissues revealed only crocidolite fibers present. One case of asbestosis was diagnosed from workers at the Nottingham factory, and two from Preston, but there are no cases concurrent with mesothelioma. (6 refs.)

- 77-3540 **Problems and Perspective in Epidemiological Study of Occupational Health Hazards in the Rubber Industry.** (Eng.) Mancuso, T. F. (Dept. Industrial Environmental Health Sciences, Graduate Sch. Public Health, Univ. Pittsburgh, Pittsburgh, PA 15261) *Environ Health Perspect* 17: 21-30; 1976.

Difficulties inherent to the epidemiological study of occupational health hazards in the rubber industry are discussed, along with data concerning the company-to-company occurrence of cancers of the gallbladder, bile ducts, lung, brain, and nervous system in the workers. The occurrence of these cancers in rubber workers generally exceeds that in the general population. (8 refs.)

- 77-3541 **Chronic Diseases in the Rubber Industry.** (Eng.) Tyroler, H. A. (Sch. Public Health, Univ. North Carolina, Chapel Hill, NC 27514) Andjelkovic, D.; Harris,

R.; Lednar, W.; McMichael, A.; Symons, M. *Environ Health Perspect* 17: 13-20; 1976.

Mortality data for the period 1964-1972 for over 10,000 workers, aged 40-64, and for about 5,000 retirees, aged 65-84, of two large US rubber and tire manufacturing companies were analyzed. These data consistently demonstrated excesses of deaths attributed to leukemia (standardized mortality ratio, SMR, range 100-315), lymphosarcoma (SMR range 108-251), stomach cancer (SMR range 111-219), cancer of the large intestine (SMR range 102-123), and cancer of the prostate (SMR range 110-183). Although no overall excess in lung cancer deaths was observed, there was a pattern of increased deaths in certain work areas, such as curing departments. Morbidity studies disclosed excesses of chronic pulmonary diseases, with evidence of an interactive effect in the association of work and smoking histories with pulmonary disability retirement. (12 refs.)

- 77-3542 **Occupational Disease in the Rubber Industry.** (Eng.) Peters, J. M. (Harvard Sch. Public Health, Boston, MA 02115) Monson, R. R.; Burgess, W. A.; Fine, L. J. *Environ Health Perspect* 17: 31-34; 1976.

Mortality patterns in a large cohort of rubber workers were studied and workers exposed to curing fumes, processing dusts, and industrial talc were examined to determine the effects of potentially hazardous materials. With the exception of cancer, there was no strong indication that there were excess deaths from any specific cause among 25,000 past and current employees from one rubber plant. Gastrointestinal (especially stomach) cancer was excessive in processing workers and lung cancer was excessive in curing workers. Leukemia and bladder cancer were increased generally. A respiratory disease study was conducted of 121 men exposed to tire-curing fumes, 65 men working in compounding, and 80 men exposed to talc. All three groups had an increase in respiratory diseases that was related to intensity and duration of exposure. Talc workers had a clear increase in respiratory morbidity at dust levels considerably below those considered safe for occupational exposures. (7 refs.)

- 77-3543 **Cancers of the Lung and Nasal Sinuses in Nickel Workers: A Reassessment of the Period of Risk.** (Eng.) Doll, R. (Dept. Regius Professor Medicine, Radcliffe Infirmary, Oxford, England) Mathews, J. D.; Morgan, L. G. *Br J Ind Med* 102:105; 1977.

Men who had been employed in a nickel refinery in South Wales on at least two occasions 5 yr or more apart before 1945 were followed up, and mortality rates from different causes were related to the date of first employment. For the

967 employees as a whole, the risk of death from carcinoma of the nasal sinus was 300-700 times the national av if they started work before 1920 and about 100 times the national av if they started work between 1920 and 1925. No deaths were attributed to nasal sinus cancer in men who started after 1924, but one man who started in 1929 developed a cancer of the ethmoid sinus. For men who started work before 1920, the mortality from lung cancer was 6-111 times the national av. The risk declined to 5.2, 2.5, and 1.5 times the national av for men who started in 1920-24, 1925-29, and 1930-34. Previous studies had suggested that the occupational risk of lung and nasal sinus cancer had been eliminated by 1925. These findings indicate that the risk persisted until 1930, which accords better with the temporal changes in the process. (12 refs.)

- 77-3544 **Mortality and Cancer Morbidity in a Group of Swedish VCM and PCV Production Workers.** (Eng.) Byren, D. (Kema Nord, 85013 Sundsvall, Sweden) Engholm, G.; Englund, A.; Westerholm, P. *Environ Health Perspect* 17: 167-170; 1976.

Mortality and cancer morbidity data are presented from studies of 750 workers employed in a Swedish vinyl chloride (VC)/poly(vinyl chloride) plant since 1940. Observed/expected deaths from various causes were: 2/0.33 (brain cancer), 3/1.78 (lung cancer), 4/0.97 (cancer of liver/pancreas, including 2 angiosarcomas of the liver), 28/18.26 (total circulatory disease), 6/3.02 (cerebrovascular disease), 15/9.15 (myocardial infarction), and 5/1.48 (cerebral hemorrhage). Observed/expected morbidities were: 3/1.30 (lung cancer) and 2/0.48 (cancer of liver/pancreas). The excess of cancers of the liver/pancreas increased with latency time (time between first employment and end of follow-up). The possible etiology of the cardiovascular deaths is discussed. (7 refs.)

- 77-3545 **Bladder Cancer: Possible New High-risk Occupation (Letter to Editor).** (Eng.) Wigle, D. T. (Bureau Epidemiology, Lab. Centre for Disease Control, Health and Welfare Canada, Ottawa, Canada K1A 0L2) *Lancet* 2(8028): 83-84; 1977.

Incidence and mortality rates from a Canadian study indicated that workers in the aluminium refining industry may be at risk for the development of bladder cancer. Polycyclic hydrocarbons given off in the refining process may be the carcinogenic agents. (6 refs.)

- 77-3546 **Leukaemia in Benzene Workers.** (Eng.) Infante, P. F. (Industry-wide Studies Branch, Div. Sur-

veillance, Hazard Evaluations and Field Studies, Natl. Inst. Occupational Safety and Health, Center for Disease Control, Cincinnati, OH 45202) Wagoner, J. K.; Rinsky, R. A.; Young, R. J. *Lancet* 2(8029): 76-78; 1977.

A follow-up study of workers exposed to benzene between 1940 and 1949 indicated a 5-fold excessive risk of all leukemias and a 10-fold excess of deaths from myeloid and monocytic leukemias in the workers as compared to the controls. This is a conservative estimate since 25% of the workers were not contacted. (22 refs.)

- 77-3547 **Clinical Studies of Styrene Workers: Initial Findings.** (Eng.) Lorimer, W. V. (Environmental Sciences Lab., Dept. Community Medicine, Mount Sinai Sch. Medicine, City Univ. New York, New York, NY 10029) Lilis, R.; Nicholson, W. J.; Anderson, H.; Fischbein, A.; Daum, S.; Rom, W.; Rice, C.; Selikoff, I. J. *Environ Health Perspect* 17: 171-181; 1977.

A clinical survey was made of 494 workers at a styrene manufacturing, polymerization, and polystyrene extrusion facility. Styrene was confirmed to be an irritant of the upper respiratory tract; it may also be an irritant of the lower respiratory tract. Thus, 30% of the nonsmoking workers had an FEV<sub>1</sub>/FVC ratio (forced expiratory volume at 1 sec/forced vital capacity) of <75%, and 12% of the highly exposed workers (compared to 4% of the slightly exposed workers) had repeated episodes of wheezing and/or tightness in the chest. Serum  $\gamma$ -glutamyl transpeptidase levels were higher among workers more highly exposed, which suggests some impairment of liver function. Hematological findings did not provide any evidence of significant marrow depression. There was no evidence of the characteristic changes associated with exposure to vinyl chloride, such as Reynaud's syndrome, acro-osteolitis, clinical hepatic dysfunction, or skin changes. No overt neoplasms were found. (37 refs.)

- 77-3548 **Registration of Data Concerning Cancers Possibly Produced by Air Pollution.** (Eng.) Clemmesen, J. (Cancer Registry, Strandboulevarden 49, 2100 Copenhagen O, Denmark) *IARC Sci Publ* (16): 159-165; 1977.

The requirements of a cancer registry for collecting data on cancers caused by air pollution are discussed. A registry should contain information for full identification of persons decades later. It may also be desirable to register spouses because of contamination of homes, eg, from work clothes. Occupations should be well-defined and information on the smoking of tobacco should be accessible, to evaluate risk on an individual and a general basis. The geographical area to be covered should be decided initially, and full information should be collected on populations, distributed by sex and



age. The best basis for analysis of a possible oncogenic influence of atmospheric pollution is a local cancer registry. The most troublesome part of such a study is the provision of control data on a scale sufficient for adequate comparison with the study area. There has been no evidence yet that lung cancer is produced by general air pollution, in areas where tobacco smoking is not practiced. Developments in applied chemistry, however, may soon result in evidence that people are exposed to oncogenic factors in the air. (3 refs.)

- 77-3549 **Chemical Composition and Potential "Genotoxic" Aspects of Polluted Atmospheres.** (Eng.) Sawicki, E. (Lab. Measurements Res. Section, Environmental Protection Agency, Research Triangle Park, NC 27711) *IARC Sci Publ* (16): 127-156; 1977.

The chemical composition of the gaseous, vapor, and particulate phases of the atmosphere is reported in terms of background, urban, and highly polluted levels. The gaseous phase of the atmosphere includes potentially dangerous agents such as nitrous compounds (which can react to form nitrosamines), alkenes, sulfur dioxide, ozone, formaldehyde and halocarbon compounds. At high concentrations, most of the vapor-phase compounds (such as hydrocarbons, alkene oxides, halocarbon compounds, peroxyacyl nitrates and phenols, nitrosamines, chloroalkyl ethers, p-dioxane and azarenes, and aldehydes) act as ciliotoxic and mucus-coagulating agents, breaking down the defenses of the respiratory system. Particulate sulfates and sulfites are mutagenic, and sulfites are also considered potential precarcinogens. All aromatic amines must be considered carcinogenic precursors, along with olefinic hydrocarbons, acids, and other analogous unsaturated derivatives in the particulate phase. Many of the atmospheric polycyclic aromatic hydrocarbons (PAH) are carcinogenic, including benzo(a)pyrene, benzo(a)anthracene, benzo(e)pyrene, benzo(a)fluoranthene, and dibenzo(a,i)pyrene. Cocarcinogens that augment PAH carcinogenicity include acetone, carbon tetrachloride, and ozone. Asbestos, which is also found in particulate form, produces an increased risk of mesotheliomas and lung cancer in humans. (187 refs.)

- 77-3550 **Environmental Load in the Federal Republic of Germany and Its Importance for Health.** (Ger.) Schlipkoter, H. W. (Dusseldorf, W. Germany) von Bogdandy, L.; Henkel, S.; Vettebrodt, K. H. *Stahl Eisen* 97(2): 61-69; 1977.

A review of the effects of West Germany environmental protection measures on air quality includes a comparison of measured and allowable values for dust, SO<sub>2</sub>, and other pollutants, and data on mortality due to environmentally related diseases. Further, epidemiological research is essential to pro-

vide protection from environmental pollutants. Data are also given graphically in 16 figures. (61 refs.)

- 77-3551 **Aviation and Environmental Benzo(a)pyrene Pollution.** (Eng.) Shabad, L. M.; Smirnov, G. A. *In: Environmental Pollution and Carcinogenic Risks.* (Lyon: International Agency for Research on Cancer): IARC Scientific Publications No. 13, INSERM Symposia Series Vol. 52, pp. 53-60; 1976.

The environmental carcinogenic pollution effect of benzo(a)pyrene (BP) in exhaust gases of turbo-jet and turbo-prop engines was tested by applying soot samples containing 0.1% BP to the interscapular skin of Fi(C<sub>3</sub>,b<sub>1</sub>,XCBA) mice. The pollution effect of BP was also analyzed in soil, vegetation, and snow samples taken from near an airport. The BP content in soil at the end of the runway comprised 64.3 and 48.7 µg/kg, which was due to the maximum working regime of the engine when it takes off. BP pollution of vegetation varied from 21.3 to 5.4 µg/kg for plants and from 7.0 to 3.1 µg/kg for the roots of these plants. BP content in snow was greater than that in soil. It was calculated that an aircraft engine discharges 4 mg BP/min, which amounts to 2 kg/yr. The concentration of carcinogenic hydrocarbons in aircraft exhausts was found to be dependent on the working regime of the engine and on the character of fuel combustion. (16 refs.)

- 77-3552 **Motoring and Cancer Risk.** (Ger.) Knutti, R. (Institut für Toxikologie der ETH und der Universität Zurich, Schorenstrasse 16, CH-8603 Schwerzenbach, Switzerland) Schlatter, C. *Schweiz Med Wochenschr* 107(9): 312-315; 1977.

After a critical review of previous findings of Blumer on the high cancer death rate among persons residing near well-traveled highways and after study of the Swiss statistics for 1959-1970, the conclusion that the lead content of auto exhaust is responsible is contested. Those persons who moved away from the highways constituted a younger age group, and there was no significant difference in incidence between EDTA-treated and untreated persons when the age structure of the two groups was considered. The occurrence of 55/231 deaths in the highway residents when 46 were expected was not significant statistically, but the number of these deaths due to cancer, 25/55, is more than might be expected by chance. Since regional differences affect the level of cancer mortality, it is suggested that similar studies be made in other regions. (12 refs.)

- 77-3553 **Fluoridation and Cancer: Age-Dependence of Cancer Mortality Related to Artificial Fluori-**

lation. (Eng.) Yiamouyiannis, J. (Natl. Health Federation, Delaware, OH 43015) Burk, D. *Fluoride* 10(3): 102-125; 1977.

The cancer death rates (CDR) of residents of fluoridated and nonfluoridated cities were analyzed in detail to determine to what extent the net increase (approx 8.5/100,000 from 1952 to 1969) in fluoridated cities could be attributed to age, race, or sex. The 10 largest fluoridated (since 1952) cities and the 10 largest nonfluoridated (as of 1969) cities were taken as the experimental and control groups, respectively. In 1953, the cancer death rate for all these cities was 155/100,000. Between 1952 and 1969, there was no significant fluoridation-linked increase in CDR in populations 0-24 and 25-44 yr old. In those 45-64 yr old, there was a fluoridation-linked increase in CDR of 15/100,000 ( $p < 0.02$ ), and in populations  $> 65$  yr old, there was an increase of 35/100,000 ( $p < 0.05$ ). Changes in the racial or sex distributions of the fluoridated and nonfluoridated populations had no effect on the fluoridation-linked increase in CDR. The values of the fluoridation-linked increases are believed to be low, since the increase in CDR in fluoridated vs nonfluoridated cities does not appear to be a linear function of time. Also, because of the movement of people and fluoride-containing food products in and out of the cities studied, the observed effects were probably diluted. (38 refs.)

**77-3554 Carcinogens in Estuaries, Their Monitoring and Possible Hazard to Man.** (Eng.) Stich, H. F.; Acton, A. B.; Dunn, B. P. In: *Environmental Pollution and Carcinogenic Risks*. (Lyon: International Agency for Research on Cancer): IARC Scientific Publications No. 13, INSERM Symposia Series vol. 52, pp. 83-94; 1976.

The feasibility of using bottom-dwelling flatfish populations and their skin tumors for examining the chemical carcinogens in subtidal or intertidal waters is examined. A trial procedure on the skin of lemon sole (*Parophrys vetulus*) along the coast of British Columbia and Washington State showed that tumor prevalences varied from 0.0% to 58.6%. The higher values were found adjacent to cities. A study of papillomas on crested flounders (*Limanda schrenki*) in two locations in Japan, one free of industrial contamination and one containing heavy deposits of peat moss, showed an occurrence of papillomas about 24 times greater in the latter location than in the former. To complement this method of monitoring carcinogens indirectly by their effect on an indicator species, a direct laboratory method is developed by establishing a 'carcinogen index' based on measurements of benzo[a]pyrene (BaP) in water bottom sediments and in the common mussel (*Mytilus edulis* or *Mytilus californianus*). The *Mytilus californianus* was the most able, since it is widely distributed, readily sampled, nonmigratory, and adaptable to laboratory experiments. Using this method, docks and wharfs appear to show a notable elevation in BaP, thought to be the result of contamination by creosote. A three-tier assessment of muta-

genic hazards is presented: the first tier is the short-term in vitro test; the second tier is the fish-tumor system; and the third is the detection of selected key compounds such as the B(a)P analysis in mussels. (24 refs.)

**77-3555 Tumor Growth Patterns in Multiple Myeloma.** (Eng.) Hokanson, J. A. (Cancer Center, Room 226, Basic Science Building, Univ. Texas Medical Branch, Galveston, TX 77550) Brown, B. W.; Thompson, J. R.; Drenwinko, B.; Alexanian, R. *Cancer* 39(3): 1077-1084; 1977.

Serial changes in tumor mass were evaluated in 61 patients with multiple myeloma who had received intermittent courses of melphalan-prednisone until death. The variations in the kinetics of tumor reduction and relapse could be explained by a mathematical model based on two cell populations, one sensitive to and one resistant to chemotherapy. For all responding patients, the median tumor halving time was 1.3 mo and the median doubling time was 2.9 mo. The duration of a constant tumor mass during remission was brief in most patients. A larger fraction of resistant cells prior to therapy was associated with a slower tumor doubling time during relapse. With a constant fractional reduction of sensitive cells and a tumor halving time of  $\leq 1$  mo, all cells sensitive to alkylating agents would be eliminated with 3 yr of uninterrupted intermittent therapy. (25 refs.)

**77-3556 Hepatomas in Marine Fish from an Urban Estuary.** (Eng.) McCain, B. B. (Dept. Pathology, Sch. Medicine, Univ. California, Davis, CA 95616) Pierce, K. V.; Wellings, S. R.; Miller, B. S. *Bull Environ Contam Toxicol* 18(1): 1-2; 1977.

The incidence of hepatoma in *Parophrys vetulus* (English sole) in the Duwamish River estuary in Seattle, Washington was 32%. Polychlorinated biphenyls are suspected as the cause. (1 ref.)

\* (Rev): 77-3008, 77-3009, 77-3010, 77-3011, 77-3012, 77-3013, 77-3014, 77-3015, 77-3016, 77-3018, 77-3020, 77-3021, 77-3022, 77-3029, 77-3037, 77-3047, 77-3048, 77-3050, 77-3051, 77-3052, 77-3053, 77-3054, 77-3055, 77-3056, 77-3057, 77-3058, 77-3059, 77-3060.

\* (Chem): 77-3100, 77-3119.

\* (Immun): 77-3330, 77-3331.

\* (Path): 77-3349, 77-3364, 77-3368, 77-3371, 77-3393, 77-3399, 77-3430, 77-3482, 77-3487.



## MISCELLANEOUS

- 77-3557 **Growth Behavior of Ascites Tumor Cells in Three-dimensional Agar Culture.** (Eng.) Sato, H. (Dept. Oncology, Res. Inst. Tuberculosis, Leprosy and Cancer, Tohoku Univ., Sendai, Japan) Goto, M.; Hosaka, S. *Tohoku J Exp Med* 122(2): 155-160; 1977.

The invasive growth of ascites tumor cells in vitro was studied with the use of a three-dimensional agar matrix into which the tumor cells migrated or infiltrated. Seventeen tumor strains were examined. These tumors grow in ascites as isolated cells (single-cell strain) or in aggregates of different cell numbers (island-type strain). Plating efficiencies of these cells were variable, depending on the tumor strain. Cells of the cultured line showed almost 100% plating efficiency irrespective of the number of cells inoculated. Tumor cells formed three types of colonies: solid type, mushroom-shaped and disk-shaped. These three colonies were observed in all the tumor strains tested, depending on their position in the agar layer. Single-cell strains tended to form loose colonies in which cell contact was loose and cells were easy to liberate from the periphery of the colony. Island-type strains had a tendency to form packed colonies in which cell contact was tight. The growth behavior of ascites tumor cells in agar culture correlated well with their growth in vivo. (8 refs.)

- 77-3558 **Selective Growth of Transformed Cell Lines by Rat Liver Perfusate.** (Eng.) Schuler, M. F. (Dept. Medicine, Milton S. Hershey Medical Center, Pennsylvania State Univ., Hershey, PA 17033) Jefferson, L. S.; Gorodecki, J.; Glaser, R.; Dietz, J.; Lipton, A. *Cancer Res* 37(6): 1662-1665; 1977.

An investigation was made of the effects of rat liver perfusate on the in vitro growth of various transformed cell lines and their nontransformed counterparts. After 5 days of incubation with perfusate, cell lines 3T12-NY (a spontaneous fibroblast transformant), NQ-T-1 (a chemically transformed fibroblast line), W-8 (a chemically transformed epithelial rat liver cell line), and H-50 (a simian virus 40-transformed hamster fibroblast line) all exhibited significant increases in cell growth over controls. Their respective nontransformed counterparts, 3T3 Cl 42, A31-714, K-16, and HEF, were not stimulated to the same extent. The virally transformed mouse fibroblast line, SV3T3, exhibited a 27-fold increase in growth; however, 3T3, Py3T3 (polyoma-transformed 3T3 cells), SV-Fl-101 (a flat revertant line), and SV-Py-3T3 cells were not responsive. It is suggested that rat liver perfusate contains a macromolecular species able to stimulate the in vitro growth of neoplastic cells selectively and that the liver may play a role in supporting neoplasia in vivo. (18 refs.)

- 77-3559 **The Control of Human WI-38 Cell Proliferation by Extracellular Calcium and Its Elimination by SV-40 Virus-induced Proliferative Transformation.** (Eng.) Boynton, A. L. (Animal and Cell Physiology Group, Div. Biological Sciences, Natl. Res. Council Canada, Ottawa, Canada K1A 0R6) Whitfield, J. F.; Isaacs, R. J.; Tremblay, R. *J Cell Physiol* 92(2): 241-248; 1977.

DNA synthesis and cell multiplication were examined in WI-38 human diploid fetal lung cells incubated in fetal bovine serum and Eagle's basal diploid medium containing free calcium at concentrations between 0.00 and 1.25 millimoles (mM). The proliferative activity of WI-38 cells in sparse cultures depended on the extracellular concentration of free (or physiologically available) calcium. Cultivation in a medium with a calcium concentration of 0.1 mM or less gradually, but reversibly, arrested their proliferative development in the prereplicative G<sub>1</sub> phase of the cell cycle. In contrast to normal WI-38 cells, cells transformed by simian virus 40 and plated at the same density were unaffected by variations in the extracellular calcium concentration. These cells retained their capacity for DNA synthesis indefinitely in medium containing  $\leq 0.1$  mM calcium. Moreover, they accumulated protein and multiplied rapidly in the presence of 0.01 mM free calcium, a concentration that was too low to support the growth and multiplication of nontransformed cells. (34 refs.)

- 77-3560 **Motility of L 5222 Rat Leukemia Cells in the Flattened State: Evidence Against Emperipolesis.** (Eng.) Haemmerli, G. (Abteilung fur Krebsforschung, Institut fur Pathologie, Birchstrasse 95, CH-8050 Zurich, Switzerland) Felix, H.; Strauli, P. *Virchows Arch (Cell Pathol)* 24(2): 165-178; 1977.

Emperipolesis is the term for the assumed penetration of living cells into other living cells. L 5222 rat leukemia cells, migrating in vitro, change from a spherical to a spread configuration when they meet flat cells and continue to move in this shape within the contours of the target cells. Whether this close cellular association corresponds to emperipolesis could not be determined by phase and interference contrast cinemicrography alone. In combination with transmission electron microscopy, however, it could be demonstrated that the compartment in which the spread leukemia cells move is not the cytoplasm of the target cells, but the narrow space created by the target cells and the underlying glass surface. Thus, emperipolesis can be ruled out for L 5222 leukemia cells. On this basis the reported observations on emperipolesis are reviewed, and a critical attitude regarding the occurrence of emperipolesis in general is advocated. (48 refs.)

- 77-3561 Development and Characterization of Established Cell Lines from Primary and Metastatic Regions of Human Endometrial Adenocarcinoma.** (Eng.) Ishiwata, I. (Dept. Obstetrics and Gynecology, Sch. Medicine, Keio Univ., 35, Shinanomachi, Shinjuku-ku, Tokyo 160, Japan) Nozawa, S.; Inoue, T.; Okumura, H. *Cancer Res* 37(6): 1777-1785; 1977.

The properties of two cell lines, SNG-P and SNG-M, that were established, respectively, from the primary and metastatic regions of a human endometrial adenocarcinoma are reported. The lines have been maintained in vitro for > 13 mo and they have been subcultivated > 65 times. The cultured cells are epithelial in shape, demonstrate a pavement arrangement, and form multilayers without contact inhibition. Most of the cells have highly indented nuclei with multiple large nucleoli, and they form desmosome contacts. The chromosomal number varies widely and shows aneuploidy, but the modal chromosome number is in the diploid range. No marker chromosome could be identified. Both cell lines could be transplanted to the cheek pouch of immunosuppressed hamsters: the resulting tumors resembled the original adenocarcinoma. The cell lines should prove useful for study of the effects of sex hormones on the growth and differentiation of endometrial carcinoma. Preliminary work shows that estradiol enhances growth rate, whereas progesterone inhibits it and also brings about specific morphological changes, including multinucleation and the shift from a pavement to a papillary cell arrangement. (28 refs.)

- 77-3562 Continuous Tissue Culture Cell Lines Derived from Chemically Induced Tumors of Japanese Quail.** (Eng.) Moscovici, C. (Tumor Virology Lab., Veterans Admin. Hosp., Gainesville, FL 32601) Moscovici, M. G.; Jimenez, H.; Lai, M. M.; Hayman, M. J.; Vogt, P. K. *Cell* 11(1): 95-103; 1977.

The establishment and properties of several cell lines derived from methylcholanthrene-induced fibrosarcomas of Japanese quail are reported. The lines, which have been maintained in continuous culture for > 3 yr, consist either of fibroblastic elements, round refractile cells, or polygonal cells. They demonstrate transformed characteristics in agar colony formation and hexose uptake, and most are tumorigenic. Their cloning efficiency in plastic dishes is not increased over that of normal quail fibroblasts. They do not produce endogenous avian oncoviruses, and they fail to complement the Brian high titer strain of Rous sarcoma virus. Those tested lack the p27 protein of avian oncoviruses. Most of the cell lines are susceptible to Subgroup A avian sarcoma viruses but relatively resistant to Subgroups C, E and F viruses, compared to normal quail embryo fibroblasts. The properties of these lines make them potentially useful tools in virology and cell genetics. (30 refs.)

- 77-3563 Characterization of a New Transitional Cell Carcinoma Line (Meeting Abstract).** (Eng.)

Moore, G. E. (Denver General Hosp., Denver, CO 80204) Swanson, T. L.; Morgan, R. T.; Quinn, L. A. *In Vitro* 13(3): 173; 1977. (no refs.)

- 77-3564 Separation of Lymphocytes and Mast Cells from the Furth Transplantable Mast Cell Tumor in an Isokinetic Gradient of Ficoll in Tissue Culture Medium.** (Eng.) Pretlow, T. P. (Dept. Pathology, Univ. Alabama Medical Center, Birmingham, AL 35294) Glover, G. L.; Pretlow, T. G. *Cancer Res* 37(2): 578-584; 1977.

Cell suspensions of the transplantable Furth murine mast cell tumor were separated both by velocity sedimentation in an isokinetic gradient and by isopycnic sedimentation. Prior to separation, the tumor cell suspension contained approx 60.3% malignant mast cells, 9.8% lymphocytes, 4.3% granulocytes, 1.7% macrophages, 0.6% unidentified cells, and 22.8% RBC. After either isokinetic or isopycnic sedimentation, > 97% of the nucleated cells in the purest modal fraction were malignant mast cells. Velocity sedimentation in the isokinetic gradient offered several advantages over isopycnic separation: the cells are exposed to a lower centrifugal force for a shorter period of time, a much larger proportion of mast cells were in the highly purified zone of the gradient, and lymphocytes were more highly purified (88.9% of the nucleated cells). Granulocytes and macrophages were purified more than eightfold over the nucleated cells in the starting sample suspension. The purified cells from this tumor offer the opportunity to study the interactions between highly purified, easily identified malignant cells and cells that may participate in the host defense against cancer. (45 refs.)

- 77-3565 Experimental Induction of Malignant Tumors in the Urinary Tract of the Rabbit.** (Ger.) Hofstetter, A. (Stadt. Oberarzt, Urol. Univ.-klinik, Thalkirchner Strasse 48, 8 Munchen 2, W. Germany) Staehler, G.; Keiditsch, E. *Fortschr Med* 95(6): 346-347; 1977.

Suspensions of the Brown-Pearce carcinoma are useful for producing bladder tumors in rabbits after injection into the wall of the exposed, but not opened, bladder. The advantages are rapid tumor growth and the possibility of observation by endoscopy and radiography. This tumor pattern is currently being used to examine the effect of different laser beams on experimental bladder tumors. (6 refs.)

- 77-3566 Adaptation of an Automatic Bacterial Colony Counter for Measuring Lung Tumor Growth in Mice.** (Eng.) Filardi, M. J. (Kidney Disease Inst., Div. Lab. and Res., New York State Dept. Health, Albany, NY 12208) Lininger, L.; McKneally, M. F. *Cancer Res* 37(part 1): 2726-2728; 1977.

An automatic bacterial colony counter was used to detect and



quantitate tumor growth in the lungs of C<sub>3</sub>H/HeJ mice. The counter was not as acute for lungs with few tumors as it was for those with many tumors. Counting tumor foci by eye is suggested for the former. (2 refs.)

77-3567 **An Analysis of Mammalian Cell Adhesion (Meeting Abstract).** (Eng.) Magilen, G. H. (Univ. California, Berkeley, CA 94720) *Diss Abstr Int [B]* 37(9): 4284-4285; 1977. (no refs.)

77-3568 **Interaction of Concanavalin A with Differentiated and Undifferentiated Murine Neuroblastoma Cells.** (Eng.) Rosenberg, S. B. (Dept. Biochemistry and Biophysics, Sch. Medicine Univ. Pennsylvania, Philadelphia, PA 19174) Charalampous, F. C. *Arch Biochem Biophys* 181(1): 117-127; 1977.

Tritiated acetyl-concanavalin A (<sup>3</sup>H-Con A) was used to examine the kinetics of Con A binding to differentiated (monolayer-cultured) and undifferentiated (spinner-cultured) murine neuroblastoma cells (clone neuro 2-A). The distribution of the Con A binding sites in the plasma membrane had been shown to be continuous in the differentiated cells and "patchy" in the undifferentiated cells. The binding of Con A at 0°C gave sigmoidal saturation curves in both instances: cooperative binding (Hill) constants were 1.75 for differentiated and 1.36 for undifferentiated cells. The maximum numbers of Con A molecules bound per cell were  $2.3 \times 10^7$  and  $3.4 \times 10^7$ , and the apparent rate constants were 6.68 and  $6.13 \times 10^2 \text{ M}^{-1} \text{ sec}^{-1}$  for the differentiated and undifferentiated cells, respectively. The differences in the kinetic behavior of Con A binding to its receptors may reflect differences in the structure or organization of the receptors between the differentiated and undifferentiated cells. These differences may be relevant to differentiation. Con A bound to spinner-cultured cells did not dissociate spontaneously over a period of 1 hr at 0°C, but it did dissociate rapidly after addition of  $\alpha$ -methyl-D-mannopyranoside. This sugar-induced dissociation of the Con A was not accompanied by shedding or inactivation of the Con A binding sites. (52 refs.)

77-3569 **The Biosynthesis of Methionine in Normal and SV-40-transformed Mammalian Cell Cultures (Meeting Abstract).** (Eng.) Jacobsen, S. J. (Brigham Young Univ., Provo, UT 84601) *Diss Abstr Int [B]* 37(9): 4323; 1977. (no refs.)

77-3570 **Altered Pyrimidine Transport in a Human Mammary Tumor Cell-Line (Meeting Ab-**

**stract).** (Eng.) Gray, P. N. (Dept. Biochemistry and Molecular Biology, Univ. Oklahoma Health Sciences Center, Oklahoma City, OK 73190) *Biophys J* 17(2): 189; 1977. (no refs.)

77-3571 **Mitochondrial and Microsomal Phospholipids of Morris Hepatoma 7777.** (Eng.) Reitz, R. C. (Div. Biochemistry, Sch. Medical Sciences, Univ. Nevada, Reno, NV 89557) Thompson, J. A.; Morris, H. P. *Cancer Res* 37(2): 561-567; 1977.

The phospholipids of both mitochondrial and microsomal membranes from normal liver, host liver, and the rapidly growing Morris hepatoma 7777 were isolated, separated, and quantitated. The total as well as the individual fatty acid concentrations and compositions were determined. The total phospholipids isolated from tumor mitochondria were increased by about 50% and the fatty acid compositions were markedly altered, compared with mitochondria from normal or host liver. The polyenoic acids were decreased, and there was a concomitant increase in the monoenes. When respiratory control was determined, the tumor mitochondria exhibited a significant decrease in this parameter. The tumor microsomal membrane fraction, however, contained about 50% less phospholipids than controls. The fatty acid patterns of the total as well as the individual phospholipids were similar to those observed in the mitochondria. The species of phosphatidylcholine from both membrane fractions were separated by argentation chromatography of the intact molecules. As predicted by the fatty acid compositions, the major species of the tumor was the monoenoic/dienoic fraction. The acyl coenzyme A:1-acyl glycerophosphorylcholine acyltransferases, which aid in controlling the fatty acid composition of phospholipids, were measured. The marked increase in activity of these enzymes toward the polyenoic and monoenoic fatty acids suggested that the polyenoic acids were not available for use in the resynthesis of the tumor phosphatidylcholine. (29 refs.)

77-3572 **Differences in Lipid Fluidity among Isolated Plasma Membranes of Normal and Leukemic Lymphocytes and Membranes Exfoliated from Their Cell Surface.** (Eng.) Van Blitterswijk, W. J. (Netherlands Cancer Inst., Div. Cell Biology, Amsterdam, Netherlands) Emmelot, P.; Hilkmann, H. A.; Oomenmeulemans, E. P.; Inbar, M. *Biochim Biophys Acta* 467(3): 309-320; 1977.

The lipid microviscosity of isolated plasma membranes of mouse thymus-derived ascitic leukemia (GRSL) cells and of the extracellular membranous vesicles exfoliated from these cells was determined to find out whether vesicle formation occurs from selected surface-membrane domains. The lipid microviscosity was also determined in isolated plasma membranes of normal thymocytes and extracellular membranes of thymus cell supernatants. The microviscosities were higher

in the isolated plasma membranes of thymocytes and GRSL cells than in the corresponding intact cells. Microviscosities in the extracellular membranes of thymocytes and GRSL cells were even higher. The fluidity difference between these membranes and the plasma membranes was most pronounced for the leukemic cells and was correlated with a large difference in cholesterol/phospholipid molar ratio. There was little or no difference between the lipid fluidity in intact membranes of a given type and that in liposomes made from their lipid extracts, which may indicate that the presence of glycoproteins in the membrane has no effect on the dynamics of the lipid core. It is suggested that extracellular membranous vesicles are shed from the surface of GRSL cells similar to the budding process of viruses, by selection of the most rigid parts of the host cell membrane. (34 refs.)

- 77-3573 **Effect of a High-Beef Diet on the Fetal Bacterial Flora of Humans.** (Eng.) Hentges, D. J. (Dept. Microbiology, Univ. Missouri, Columbia, MO 65201) Maier, B. R.; Burton, G. C.; Flynn, M. A.; Tsutakawa, R. K. *Cancer Res* 37(2): 568-571; 1977.

Ten volunteer subjects completed a 4-mo diet series consisting of 1 mo each of a control diet, a meatless diet, a high-beef diet, and the same control diet. Fat and fiber contents were essentially the same in all four diets, but protein content was doubled during the high-beef diet. During the fourth week on each diet, three stool specimens from each volunteer were analyzed for chemical composition and content of facultative, aerobic, and anaerobic bacteria. High beef protein consumption had little effect on the composition of the intestinal flora. There were no significant differences in total counts of facultative and aerobic or anaerobic organisms in the feces when volunteers were on meatless or high-beef diets. At the species level, when counts during the two control diets were comparable, in only three instances did the change from the meatless to a high-beef diet significantly influence the bacterial numbers. The ratio of mean counts of anaerobic to facultative and aerobic organisms was approx 15:1 during the meatless diet and 34:1 during the high-meat diet. The data indicate that animal protein consumption has little effect on the fecal bacterial profile in humans. (19 refs.)

- 77-3574 **Characterization of Amyloid-associated Peptides from Medullary Thyroid Carcinoma** (Meeting Abstract). (Eng.) Takahashi, N. (Vanderbilt Univ. Medical Sch., Nashville, TN 37232) Page, D.; Inagami, T.; Tashjian, A. *Fed Proc* 36(3): 369; 1977. (no refs.)

- 77-3575 **Human Liver Ferritin: Tumor-specific Alterations in Structure and Antigenicity** (Meeting

Abstract). (Eng.) Snyder, D. (Dept. Medicine, Harvard Medical Sch., Boston, MA) *Gastroenterology* 72(5/Part 2): 1189; 1977. (no refs.)

- 77-3576  **$\alpha_1$ -Antitrypsin: A Tumor Protein Marker in Oral Contraceptive-Associated Liver Tumors** (Meeting Abstract). (Eng.) Palmer, P. E. (Dept. Pathology, Tufts Univ. Sch. Medicine, Boston, MA) Christopherson, W. M.; Wolfe, H. J. *Lab Invest* 36(3): 347-348; 1977. (no refs.)

- 77-3577 **Structure-Function Relations in Phosphorylcholine-binding Mouse Myeloma Proteins.** (Eng.) Goetz, A. M. (Church Lab. Chemical Biology, Div. Chemistry and Chemical Engineering, California Inst. Technology, Pasadena, CA 91125) Richards, J. H. *Proc Natl Acad Sci USA* 74(5): 2109-2112; 1977.

Binding site interactions between the phosphorylcholine (P-Cho)-binding mouse myeloma proteins TEPC 15, W3207, McPC 603, MOPC 167, and MOPC 511 and the isotopically substituted hapten phosphoryl-[methyl- $^{13}\text{C}$ ] choline were investigated using  $^{13}\text{C}$  and  $^{31}\text{P}$  nuclear magnetic resonance (NMR) spectroscopy. Each protein exhibited a unique NMR pattern, but extensive similarities in chemical shift parameters upon binding of hapten to immunoglobulin suggest a significant degree of conservation of important hapten-binding site interactions. This conservation is, however, not absolute, and deviations were observed in subregions of the binding pockets of MOPC 1167 and McPC 1603. The hapten-binding site interactions correlated with the binding affinities of these proteins for various haptens. Among these proteins, changes in binding properties can be accounted for largely by differences in the heavy chains, which appear to play a dominant role in determining specificity for P-Cho. The fundamental significance of the pairing of similar heavy chains with at least three distinct types of light chains is not clear, but hybridization experiments suggest that high affinity for P-Cho requires specific association between heavy and light chains. (22 refs.)

- 77-3578 **Transfer Ribonucleic Acid Modification and Its Relationship to Tumorous and Non-Tumorous Plant Growth** (Meeting Abstract). (Eng.) Jones, L. H. (Univ. North Carolina at Chapel Hill, Chapel Hill, NC 27514) *Diss Abstr Int [B]* 37(8): 3743; 1977. (no refs.)

- 77-3579 **Cytophotometric Determination of the DNA Content in Neoplastic Cells.** (Rus.) Petrova, A.



S. (Oncological Scientific Center, USSR Acad. Medical Sciences, Moscow, USSR) Zubrikhina, G. N. *Arkhh Patol* 39(1): 65-68; 1977.

Differences in DNA content between malignant and normal somatic cells were assayed cytophotometrically. There were 47 patients with lung cancer, 32 with gastritis accompanied by proliferating elements, 35 with cancer of the stomach, 21 with skin melanomas, 42 with malignant lymphomas and metastases to the lymph nodes, and 34 subjects with a normal gastric mucosa. Cells obtained from normal tissue (gastric mucosa, lung tissue) were predominantly diploid ( $2n$ , 84%-98%), and the amount of proliferating cells (tri- and tetraploid) was only 0.2%-5%. In the patients with benign lesions (polyposis, gastritis, gastric ulcer), the amount of diploid cells ranged from 72% to 92%, and the proliferating cells comprised 1%-18%. In the patients with malignant neoplasms, the proliferative activity ( $3n$  and  $4n$ ) ranged from 5% to 27% and 9%-40% of the cells were  $> 4n$ . The tendency toward polyploidy was more pronounced in the less-differentiated tumors. (15 refs.)

77-3580 The Effects of DNA Structure on In Vitro Transcription (Meeting Abstract). (Eng.) Botchan, P. M. (Univ. California, Berkeley, CA 94720) *Diss Abstr Int [B]* 37(9): 4436; 1977. (no refs.)

77-3581 Determination In Vitro of the Duration and Rate of DNA Synthesis In Tumor Cell Subpopulations Isolated By Ficoll Density Gradient Centrifugation (Meeting Abstract). (Eng.) Flood, O. P. (New York Univ., New York, NY 10003) *Diss Abstr Int [B]* 37(9): 4275; 1977. (no refs.)

77-3582 Distribution of DNA Repair in Chromatin (Meeting Abstract). (Eng.) Bodell, W. J. (Tumor Biology Lab., Univ. Nebraska, Lincoln, NB 68588) *Proc Am Assoc Cancer Res* 18: 118; 1977. (no refs.)

77-3583 The *dnaB* Gene Product: A Comparison of Its Role in DNA Polymerase III-Directed Replicative and X-Ray Induced Repair Synthesis (Meeting Abstract). (Eng.) Billen, D. (Univ. Tennessee-Oak Ridge Graduate Sch. Biomedical Sciences, Oak Ridge, TN 37830) Hellerman, G. R. *Biophys J* 17(2): 143; 1977. (no refs.)

77-3584 Location of Eukaryotic DNA Polymerases (Meeting Abstract). (Eng.) Foster, D. N. (Univ. California, Berkeley, Berkeley, CA 94720) *Diss Abstr Int [B]* 37(9): 4275-4276; 1977. (no refs.)

77-3585 Cation Preferences of the DNA Polymerases in Plasma from Patients with Leukemia or Breast Cancer. (Ger.) Rainer, H. (I. Medizinischen Universitätsklinik, Lazarettgasse 14, A-1090 Vienna, Austria) Moser, K. *Wien Klin Wochenschr* 89(7): 244-245; 1977.

Plasma reverse transcriptase from leukemia patients prefers the divalent cation  $Mn^{++}$  to  $Mg^{++}$  in the DNA polymerase assay. The reverse is true for the enzyme in plasma of breast cancer patients. This cation preference is analogous to that found in B- and C-type RNA tumor viruses. (4 refs.)

77-3586 Metabolic Regulation and Relationship of Endogenous Protein Kinase Activity and Steroidogenesis in Isolated Adrenocortical Carcinoma Cells of the Rat. (Eng.) Sharma, R. K. (Dept. Biochemistry, Univ. Tennessee Center Health Sciences, Memphis, TN 38163) Shanker, G.; Ahmed, N. K. *Cancer Res* 37(2): 472-475; 1977.

In rat adrenocortical carcinoma 494 cells, in contrast to normal isolated adrenal cells, 10-50  $\mu$ U of ACTH did not raise the level of cyclic AMP protein kinase activity and steroidogenesis. This indicates a lesion in the tumor adenylate cyclase system. At concentrations (0.2-10 mM) that stimulate corticosterone synthesis in normal adrenal cells, cyclic AMP and guanosine cyclic 3',5'-monophosphate had no effect on steroidogenesis in tumor cells but they stimulated protein kinase activity in a dose-dependent manner. Neither cycloheximide nor actinomycin D inhibited phosphorylation stimulation, indicating that the tumor cyclic nucleotide-dependent protein kinase activity is unrelated to steroidogenesis and is also not under transcriptional or translational control. Micromolar concentrations of cyclic AMP, in contrast to cyclic GMP, stimulated protein kinase activity. In normal cells, both cyclic nucleotides in this concentration range stimulated protein kinase without an increase in steroidogenesis. This indicates that there is an additional defect in cyclic GMP-dependent tumor protein kinase. (25 refs.)

77-3587 Regulation of Acid Proteases During Growth, Quiescence and Starvation in Normal and Transformed Cells. (Eng.) Lockwood, T. D. (Lab. Cell Biology, Salk Inst., San Diego, CA 92112) Shier, W. T. *Nature* 267(5608): 252-254; 1977.

The activity of the lysosomal protease cathepsin D was measured in nontransformed 3T3 cells, in simian virus 40 (SV40)-transformed 3T3 cells (SV3T3), and in benzo(a)pyrene-transformed 3T3 cells (BP3T3) under conditions of active growth, quiescence with adequate nutrition, and quiescence with inadequate nutrition. In nontransformed 3T3 cells, cathepsin D activity was low in growing cells, elevated 5-6 times when in cells quiescent with adequate nutrition and elevated 10-15 times in cells quiescent because of inadequate nutrition. The highly transformed SV3T3 cells, however, which had lost their ability to enter the  $G_0$  phase in response to starvation, continued to grow even under conditions of inadequate nutrition, and cathepsin D levels remained low, comparable to those in growing nontransformed 3T3 cells. In response to inadequate nutrition, BP3T3 cells, which enter  $G_0$  only under conditions of extreme serum deprivation, exhibited an intermediate degree of loss of enzyme activity compared to the 3T3 and SV3T3 cells. The pronounced elevation of lysosomal protease activities in nontransformed cells upon quiescence and starvation and the regulatory deficiency of proteases in transformed cells suggest an involvement of these enzymes in normal and altered proliferative control. (15 refs.)

**77-3588 Lysosomal Enzyme Activities in the Regenerating Rat Liver.** (Eng.) Fiszler-Szafarz, B. (Fondation Curie-Institut du Radium, Universite de Paris-Sud, Batiment 111, 91405 Orsay, France) Nadal, C. *Cancer Res* 37(2): 354-357; 1977.

The activity of four lysosomal enzymes (hyaluronidase,  $\beta$ -N-acetylglucosaminidase, acid phosphatase, and cathepsin D) was studied in aqueous extracts of the light mitochondrial fraction of regenerating male Wistar rat liver. This tissue was chosen as a model for normal cell division in vivo. In the first wave of division, 40%-50% of the cells divided synchronously. Activities were measured at 0, 9, 18 (end of  $G_1$  phase), 24 (S phase), and 30 hr (mitosis) and during regeneration, 4 and 11 days after partial hepatectomy. The activities were related to fresh tissue wt, cellular DNA, and protein content of the extracts. At 9 hr, there was a significant increase in hyaluronidase and cathepsin D activities (these two enzymes act on macromolecules);  $\beta$ -N-acetylglucosaminidase and acid phosphatase activities were only slightly increased. At the end of the  $G_1$  phase 40%-50% of the activity of all four enzymes was lost, which might indicate complete loss of activity in cells undergoing division. This depletion persisted until mitosis was complete. Four days later, there was a slow restoration of enzyme activities; after 11 days, hyaluronidase and cathepsin D exhibited about 80% of their initial activity, but  $\beta$ -N-acetylglucosaminidase and acid phosphatase regained only about 50%. These results show that the lysosomal system may play some role in cell division. (29 refs.)

**77-3589 Regulation of Tyrosine 3-Monooxygenase Activity in Pheochromocytoma Cells (Meeting Abstract).**

(Eng.) Perlman, R. L. (Dept. Physiology, Harvard Medical School, Boston, MA 02115) Chalfie, M. *Fed Proc* 36(3): 879; 1977. (no refs.)

**77-3590 Comparison Between  $Ca^{2+}$  and  $Mg^{2+}$  on Surface-Located ATPase of Intact Normal and Neoplastic Human Cells in Culture.** (Eng.) Agren, G. (Inst. Medical and Physiological Chemistry, Biomedical Center and Div. Cell Biology, Wallenberg Lab. Univ. Uppsala, Uppsala, Sweden) Ponten, J.; Ronquist, G.; Westermark, B. *Acta Physiol Scand* 98(2): 263-265; 1976.

The divalent cation dependence of ATPase activity at the surface of human glia and glioma cells was studied in cultures incubated with  $Mg^{2+}$  and/or  $Ca^{2+}$  in the presence of 250 nanomoles of ATP. The surface ATPase activity of glia cells was stimulated slightly by  $Mg^{2+}$  (40  $\mu$ moles), but not by  $Ca^{2+}$  (20  $\mu$ moles) or combinations of the two (20-40  $\mu$ moles). In glioma cells, enzyme activity was significantly higher with  $Mg^{2+}$  than with  $Ca^{2+}$ . Combinations of  $Mg^{2+}$  and  $Ca^{2+}$  did not stimulate ATPase activity more than did  $Mg^{2+}$  alone. Although the activity of  $Mg^{2+}$  stimulated ATPase was higher on the surface of glia cells than on that of glioma cells, the results do not support the hypothesis that enzyme activity in tumor cells is exclusively  $Ca^{2+}$ -dependent. (11 refs.)

**77-3591 Purification and Characterization of Thymidylate Synthetase from the Blast Cells of Patients with Acute Myelocytic Leukemia (Meeting Abstract).** (Eng.) Dolnick, B. J. (Dept. Experimental Therapeutics and Grace Cancer Drug Center, Roswell Park Memorial Inst., Buffalo, New York, NY 14263) Cheng, Y. *Fed Proc* 36(3): 774; 1977. (no refs.)

**77-3592 Regulation of HMG-CoA Reductase in HTC Cells (Meeting Abstract).** (Eng.) Beirne, O. R. (Univ. California, San Francisco, CA 94132) *Diss Abstr Int [B]* 37(9): 4435-4436; 1977. (no refs.)

**77-3593 Superoxide Dismutase Activity of Ehrlich Ascites Tumor Cells.** (Eng.) Sahu, S. K. (Radiation Res. Lab., Room 14, Medical Labs., Univ. Iowa, Iowa City, IA 52242) Oberley, L. W.; Stevens, R. H.; Riley, E. F. *J Natl Cancer Inst* 58(4): 1125-1128; 1977.

Polyacrylamide gel electrophoresis of a crude extract of Ehr-



lich ascites tumor cell homogenate produced three distinct bands of superoxide dismutase activity. The activity bands migrated approx the same distance and were inhibited by cyanide ions. Isolated mitochondria produced two bands of activity that were also inhibited by cyanide. Ethanol-chloroform treatment of the homogenate had no observable effect on these activity bands, which suggests that the cyanide-insensitive mitochondrial superoxide dismutase activity in these malignant cells was either present in concentrations below detectable levels or completely absent. Normal liver was used as a control for the detection system. (15 refs.)

- 77-3594 Adenylate Cyclase Permanently Uncoupled from Hormone Receptors in a Novel Variant of S49 Mouse Lymphoma Cells.** (Eng.) Hega, T. (Dept. Pharmacology, Univ. Virginia Sch. Medicine, Charlottesville, VA 22903) Ross, E. M.; Anderson, H. J.; Gilman, A. G. *Proc Natl Acad Sci USA* 74(5): 2016-2020; 1977.

The selection and characterization of a variant S49 mouse lymphoma clone are reported. The variant was selected from wild-type cells by growth in medium containing the  $\beta$ -adrenergic agonist terbutaline and inhibitors of cyclic nucleotide phosphodiesterase. The new variant clones have a heritable and stable alteration ( $> 100$  generations) that results in uncoupling of the hormone-binding and catalytic functions of the  $\beta$ -adrenergic receptor-adenylate cyclase system. Synthesis of cyclic AMP is not stimulated by  $\beta$ -adrenergic agonists or by prostaglandin  $E_1$ , either in intact variant cells or in membrane preparations of the clones. However, basal and NaF-stimulated activities of adenylate cyclase are normal, enzyme activity is stimulated by guanylyl-5'-yl imidodiphosphate, intact cells accumulate cyclic AMP when exposed to cholera toxin, and cell membranes possess ligand-binding activity. The molecular lesion that accounts for these properties is unknown, but these clones should serve as useful tools for study of the coupling of hormone receptor to adenylate cyclase. (21 refs.)

- 77-3595 Effect of Prostaglandin Endoperoxides and Metabolites on Bone Resorption In Vitro.** (Eng.) Raisz, L. G. (Dept. Medicine, Univ. Connecticut Health Center, Farmington, CT 06032) Dietrich, J. W.; Simmons, H. A.; Seyberth, H. W.; Hubbard, W.; Oates, J. A. *Nature* 267(5611): 532-534; 1977.

Several early products of prostaglandin arachidonic acid metabolism via the cyclo-oxygenase pathway were tested for their ability to release  $^{45}\text{Ca}$  from cultured fetal rat long bones, to elucidate their possible role in bone loss and hypercalcemia in malignancy, rheumatoid arthritis, and periodontal disease. These metabolites included the prostaglandin endoperoxides  $\text{PGG}_2$  and  $\text{PGH}_2$ ; the 13,14 dihydro derivatives  $\text{H-PGE}_2$  and

$\text{H}_2\text{-PGE}_1$ ; and the 15-keto-13,14-dihydro derivatives  $15\text{K-H}_2\text{-PGE}_2$  and  $15\text{K-H-PGF}_2\alpha$ . The endoperoxides caused a rapid transient increase in the release of previously incorporated  $^{45}\text{Ca}$  from bone, but did not stimulate prolonged resorption.  $\text{H}_2\text{-PGE}_2$  and  $\text{H}_2\text{-PGE}_1$  were almost as potent as the parent compounds.  $15\text{K-H}_2\text{-PGE}_2$  and  $15\text{K-H-PGF}_2\alpha$  were much less potent, but they did stimulate resorption at a concentration of  $10^{-5}$  M. The transient effect of  $\text{PGG}_2$  and  $\text{PGH}_2$  raises the possibility that bone tissue enzymically converts  $\text{PGG}_2$  and  $\text{PGH}_2$  to a labile product, such as thromboxane  $A_2$  or prostacyclin. (25 refs.)

- 77-3596 Quantification of Prolactin Receptors in Various Murine Mammary Tumors and the Effect of In Vivo Bound Prolactin on the  $^{125}\text{I}$ -Prolactin Radioreceptor Assay (Meeting Abstract).** (Eng.) Brooks, C. L. (Michigan State Univ., East Lansing, MI 48823) *Diss Abstr Int [B]* 37(9): 4331-4332; 1977. (no refs.)

- 77-3597 Hormone and Growth Factor Requirements for Estrogen-stimulated Growth of Rat Pituitary Tumor Cells (Meeting Abstract).** (Eng.) Sorrentino, J. M. (Univ. Texas Medical Sch., Houston, TX 77025) Sirbasku, D. A. *Fed Proc* 36(3): 366; 1977. (no refs.)

- 77-3598 Specific Estrogen Receptor in Rat and Beef Thymus (Meeting Abstract).** (Eng.) Grossman, C. J. (Dept. Physiology, Univ. Cincinnati Coll. Medicine, Cincinnati, OH 45219) Nathan, P. *Fed Proc* 36(3): 1236; 1977. (no refs.)

- 77-3599 The Role of the Combination of Dead Cells and Neutral Fats of the Body in Cancer (Letter to Editor).** (Eng.) Wales, A. (155 Ashgrove Ave., South Shields, Tyne and Wear, NE34 8BA, England) *Am J Clin Nutr* 30(5): 657-658; 1977.

It is suggested that the combination of dead cells and neutral body fats is involved in the pathogenesis of most cancers. The occurrence of stomach cancer in Japan, where ulcers and the consumption of marine fat could be of significance, is cited as an example. (2 refs.)

- 77-3600 Erythroid Precursors in Human Leukemia (Meeting Abstract).** (Eng.) Wolman, S. R. (NYU Sch. Medicine, New York, NY 10016) Horland, A. A. *Proc Am Assoc Cancer Res* 18: 192; 1977. (2 refs.)

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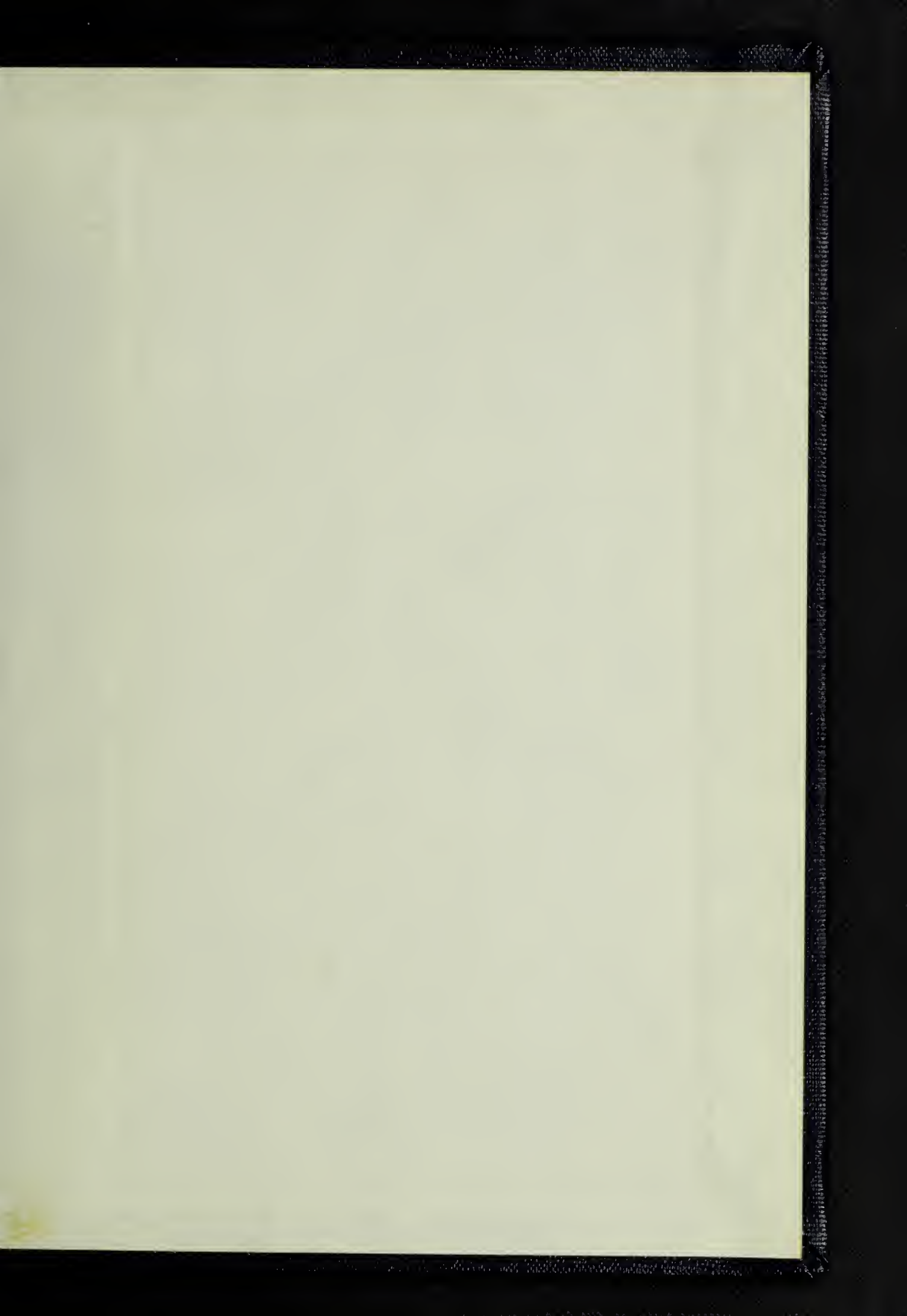














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